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ARIZONA MEDICINE

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MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO

(ISSN 0004-1556)

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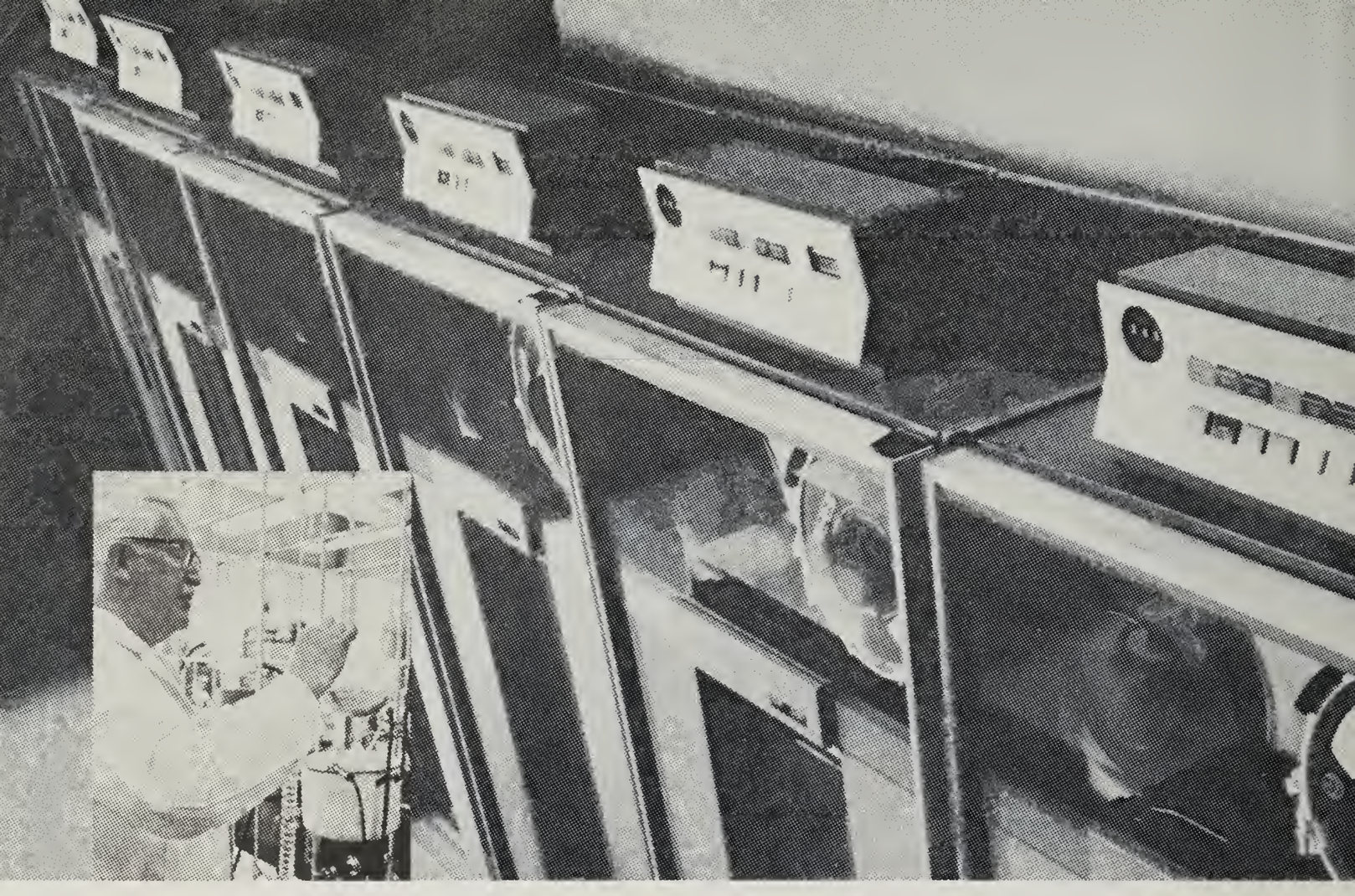
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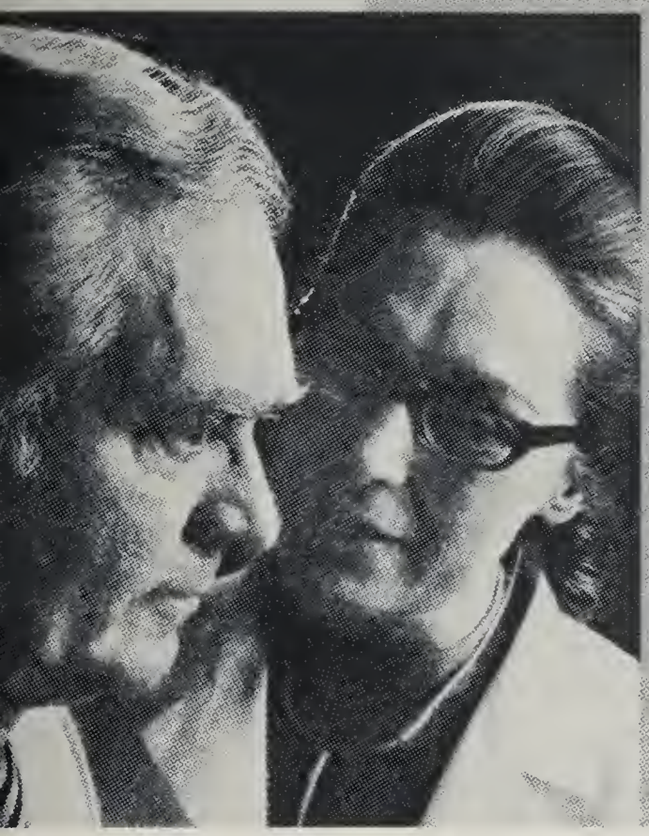
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synonymous with relief of anxiety

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- ☐ Predictable patient response
- ☐ Is used concomitantly with primary medications, such as anticholinergics and cardiovascular drugs

Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended

Contraindications: Patients with known hypersensitivity to the drug

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral—Adults.* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable

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For information on the preparation of articles write the Managing Editor, 810 W. Bethany Home Road, Phoenix, AZ 85013. (602) 246-8901.

An added complication... in the treatment of bacterial bronchitis*



Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about 2.5 percent of patients and included diarrhea (1 in 70) and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in about 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less than 1 in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGOT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [070379R]

Some ampicillin-resistant strains of Haemophilus influenzae—a recognized complication of bacterial bronchitis*—are sensitive to treatment with Ceclor.[1-6]

In clinical trials, patients with bacterial bronchitis due to susceptible strains of Streptococcus pneumoniae, H. influenzae, S. pyogenes (group A beta-hemolytic streptococci), or multiple organisms achieved a satisfactory clinical response with Ceclor.[7]

Ceclor®
cefaclor

Pulvules®, 250 and 500 mg

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae* [8]

Note: Ceclor® (cefaclor) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

References

1 Antimicrob Agents Chemother., *8*: 91, 1975
2 Antimicrob Agents Chemother., *11*: 470, 1977
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4 Antimicrob Agents Chemother., *12*: 490, 1977
5. Current Chemotherapy (edited by W Siegenthaler and R Luthy), II: 880 Washington, D.C.: American Society for Microbiology, 1978.
6 Antimicrob Agents Chemother., *13*: 861, 1978
7 Data on file, Eli Lilly and Company
8 Principles and Practice of Infectious Diseases (edited by G L. Mandell, R G. Douglas, Jr., and J E. Bennett), p 487 New York: John Wiley & Sons, 1979

Additional information available to the profession on request from Eli Lilly and Company, Indianapolis, Indiana 46285

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 No renal damage has been reported in long-term use.
- **Treat again and again if needed**
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- **Treat patients with pre-existing renal failure**
 MONISTAT* i.v. has not been reported to cause further renal damage.

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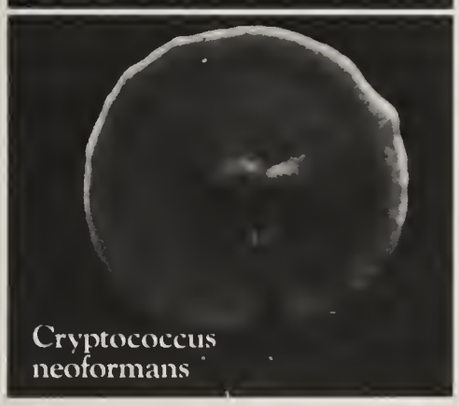
Candida albicans



Coccidioides immitis



Paracoccidioides brasiliensis



Cryptococcus neoformans

Highly effective, improvement often seen within 1-3 weeks

Patients who do not respond or develop resistance to amphotericin B have responded to MONISTAT* i.v.

Resistance does not develop

In vitro resistance of clinical isolates has not been reported during clinical trials.

Generally well tolerated by severely debilitated patients

Patients with cancer, those receiving immunosuppressive agents, and patients with serious underlying conditions, such as diabetes mellitus or renal failure.

Photos courtesy of Dubos, R.J., Bacterial and Mycotic Infections of Man, J.B. Lippincott Company, 1965 and Burrows, W., Textbook of Microbiology, W.B. Saunders Company, 19th edition.

Please turn page for Prescribing Information.



An important advance in the treatment of systemic mycoses

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(miconazole)

broad-spectrum antifungal agent

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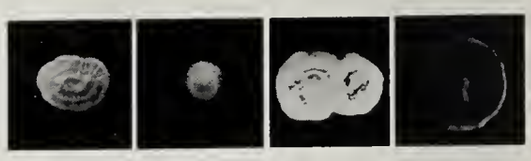
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JPI-024

Broad-spectrum antifungal agent

Monistat i.v.™
(miconazole)
for intravenous infusion



DESCRIPTION: MONISTAT i.v. (miconazole), 1- [2- (2,4-dichlorophenyl)- 2- [(2,4-dichlorophenyl) methoxyl] ethyl]-1H-imidazole, is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v. is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in-vitro antifungal activity of MONISTAT i.v. is very broad. Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis*, *Candida albicans*, *Cryptococcus neoformans*, and *Paracoccidioides brasiliensis*.

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1µg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MIC's should be determined.

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v. may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v. therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported. If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle, Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued.

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported. In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated.

DOSAGE AND ADMINISTRATION:

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended.

Organism	Dasage Range *	Durotion of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to >20
Cryptococcosis:	1200 to 2400 mg per day	3 to >12
Candidiasis:	600 to 1800 mg per day	1 to >20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to >16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children. A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium.

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because so much remains to be done

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Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

Merrell

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Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate—it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

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For prescribing information see opposite page

In G.I. therapy



Adjunctive Librax®

Each capsule contains
5 mg chlordiazepoxide HCl
and 2.5 mg clidinium Br

antianxiety/antisecretory/antispasmodic

for adjunctive therapy of duodenal ulcer* and irritable bowel syndrome*

Librax®

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide

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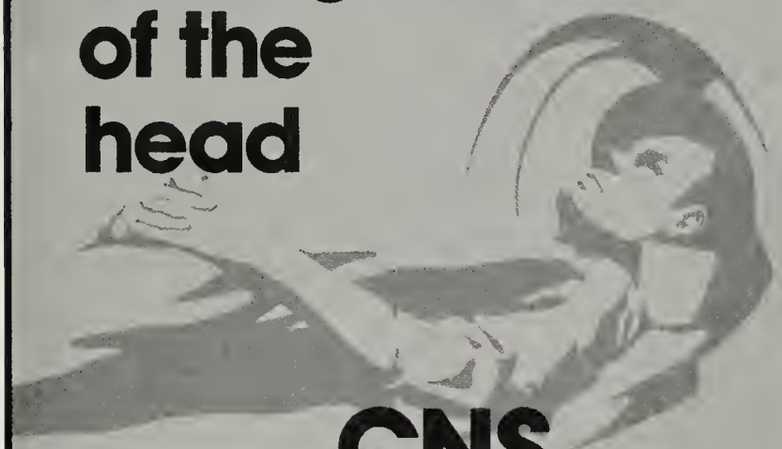
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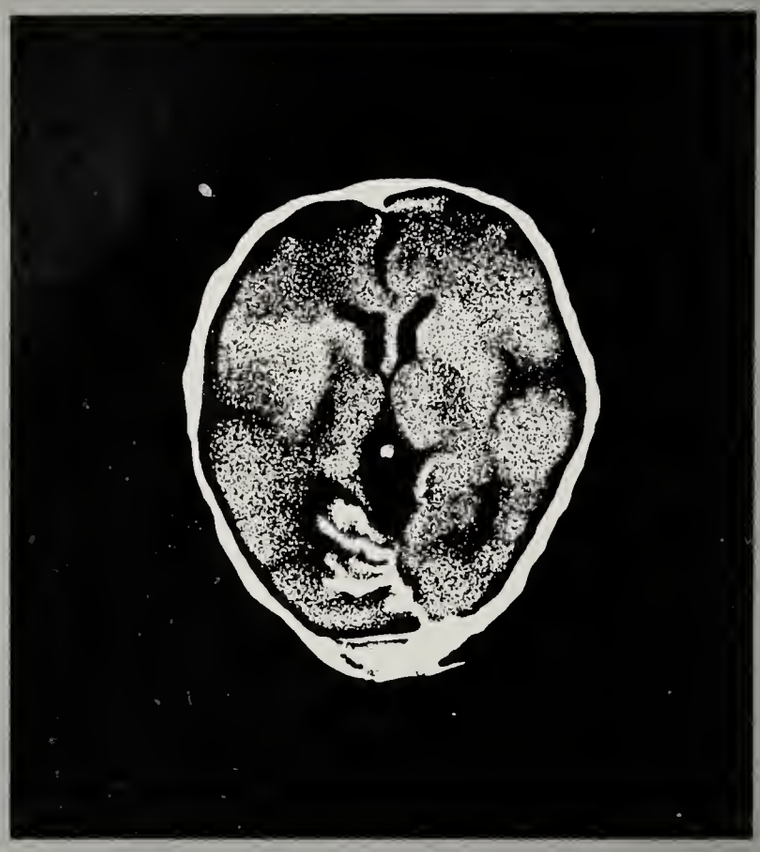
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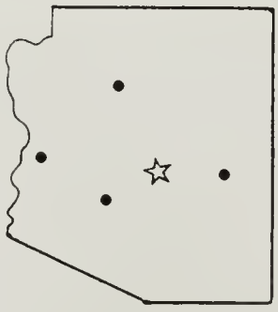


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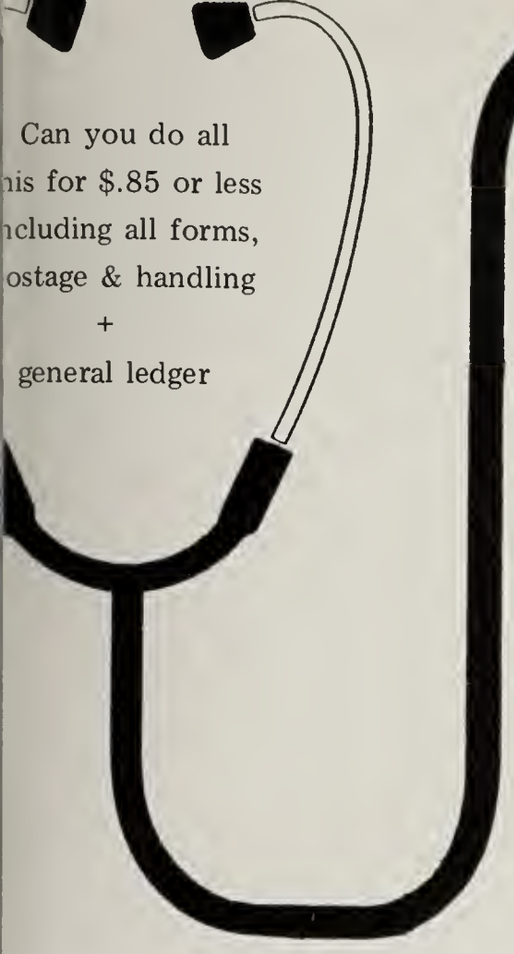
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Cardiology

Drug Interactions
In Antihypertensive and Antiarrhythmic Therapy

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Most patients with cardiovascular disease receive several drugs in order to decrease the unwanted effects from any individual agent and to increase the overall efficacy of therapy. Koch-Weser has found that in a group of 500 patients hospitalized for cardiovascular disease a median of 12 drugs is prescribed during their hospitalization.[1] It is to be emphasized that in many instances this polypharmacy is appropriate and to be encouraged in order for the patient to derive maximum benefit with the least toxicity from any individual drug. However, when a patient is treated concurrently with a large number of drugs, the possibility always exists for drug interactions. Many of the interactions seen in cardiovascular therapy are planned to take advantage of different properties of different drugs. Thus, beta-adrenergic blockers are frequently combined with diuretics and vasodilators in the treatment of moderate to severe hypertension. This combination has been shown to increase the efficacy of the individual drugs and to decrease the side effects. Similarly, a potassium sparing and a potassium losing diuretic may be combined in order to achieve adequate diuresis without change in total body potassium. These planned interactions are numerous and well understood. This paper will discuss the adverse drug interactions that can occur in cardiovascular drug therapy.

In general, drug interactions can be divided into 1) those resulting in more or less drug (or active metabolites) available at the site of action, the so-called "pharmacokinetic drug interactions," and 2) those caused by other mechanisms, the so-called "pharmacodynamic drug interactions." Both types of interactions are important.

Interactions with Antihypertensive Drugs

Diuretics

Diuretics are the most commonly used antihypertensive drugs. Pharmacokinetic interactions affecting the efficacy of the diuretic drugs in lowering

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blood pressure appear to be unimportant. As long as renal function is intact, other classes of drugs do not interfere with the diuretic action or increase the toxicity of either furosemide or the thiazides. Even probenecid, a drug that interferes with the mechanism by which furosemide reaches its tubular site of action, does not decrease the overall diuresis produced by furosemide.

Hypokalemia is a consequence of the thiazides and the "loop" diuretics, furosemide and ethacrynic acid. Whereas this usually is not a significant problem in antihypertensive drug therapy, it may be a problem in patients receiving digitalis glycosides since hypokalemia can potentiate the toxic effects of digitalis. For this reason, serum potassium should be monitored closely in patients receiving a combination of diuretic and digitalis. In a few patients, the potassium depletion and/or a direct action of thiazide diuretics may impair glucose tolerance. An occasional patient who has diabetes and is also receiving a thiazide diuretic or furosemide may require an alteration in his hypoglycemic regimen in order to keep his blood sugar controlled. Fortunately, this is an uncommon occurrence in diabetics and probably of clinical importance in less the 10% of such patients.

A potentially important side effect of thiazides is an elevation of serum cholesterol and triglyceride. The mechanism is unknown but it is possible that the increase in lipids may partially reverse the salutary effect of thiazides on blood pressure and may counteract the lipid lowering effects of hypolipidemic agents.

Sympatholytic Drugs

Clonidine

Clonidine is an antihypertensive drug that stimulates alpha-adrenergic receptors in the central nervous system. A consequence of this alpha-adrenergic receptor stimulation is a decrease in sympathetic outflow to the periphery and hence an antihypertensive effect. The tricyclic antidepressants will interfere with the antihypertensive effect of clonidine.[2] The probable mechanism of this interaction is that the tricyclic antidepressants occupy the alpha-adrenergic receptor where clonidine exerts its hypotensive effect. In experimental animals, central alpha-adrenergic blocking agents are known to decrease the effect of clonidine and probably also will decrease the effect of methyldopa. Clinically, the interaction of tricyclic antidepressants with clonidine is important, but there is very little evidence that the interaction of tricyclic antidepressants and methyldopa causes an important loss of antihypertensive effect.

Guanethidine.

Guanethidine and related drugs (bretylium, bethanidine) block periperal sympathetic nerve function. In order to be effective, guanethidine must be actively transported into sympathetic nerve terminals by the "norepinephrine pump." Drugs that inhibit the uptake of norepinephrine into adrenergic nerve endings will also inhibit the efficacy of guanethidine by blocking its entry into nerve endings. Thus, the tricyclic antidepressants are very effective in inhibiting or reversing the antihypertensive effect of guanethidine.[3] This interaction can be clinically important in severely hypertensive patients treated with guanethidine who then receive one of the several tricyclic antidepressants. Other drugs can also inhibit the norepinephrine pump, including ephedrine, phenothiazines, amphetamines, and cocaine and all of these drugs can also inhibit the antihypertensive efficacy of guanethidine. Since several over the counter preparations contain ephedrine and related drugs, this can be an important source of drug interaction and a reason for guanethidine resistance in patients.[4]

Monoamine oxidase inhibitors

Although rarely used for the treatment of hypertension, the monoamine oxidase inhibitors are still used for the treatment of depression. During treatment with monoamine oxidase inhibitors, the norepinephrine stores in nerve endings increase. In addition, the metabolism of tyramine contained in certain foods is inhibited. When such tyramine-containing foods (certain cheeses, wines, beers) are ingested by a patient receiving monoamine oxidase inhibitors, the tyramine escapes metabolism by the gastrointestinal tract and liver and gains access to the circulation. The circulating tyramine can then release the excessive stores of norepinephrine from adrenergic neurons and thereby produce a hypertensive crisis.[5] All patients receiving MAO inhibitors should be cautioned to avoid these tyramine-containing foods. It should also be noted that drugs that can release norephinephrine from nerve endings (such as indirectly acting sympathomimetic amines including metaraminol and ephedrine) may also cause a hypertensive crisis in patients receiving monoamine oxidase inhibition.

Propranolol

Pharmacokinetic interactions altering the amount of propranolol in the body are not of great clinical importance. It does appear probable, however, that treatment with a drug that can induce hepatic microsomal enzymes (such as phenobarbital) will decrease the amount of propranolol in the circulation during oral therapy. However, because the antihypertensive effects of propranolol are poorly related to the drug levels of propranolol, this does not appear to be of great clinical importance.

Pharmacodynamic interactions with propranolol can be important. Propranolol but probably not metoprolol, appears to slightly delay the recovery of blood sugar if a patient has become hypoglycemic. More importantly, all beta-adrenergic blockers can mask many of the symptoms of hypoglycemia that are related to activation of the sympathetic nervous system. Thus, patients receiving beta-adrenergic blocking drugs do not have a tachycardia, tremor, or a feeling of anxiety with hypoglycemia, although they will continue to have the mental changes and sweating.[6] In addition, propranolol administration to patients who have elevated levels of

circulating epinephrine will produce a hypertensive response.[7] Since epinephrine is released in patients with hypoglycemia, in patients with pheochromocytoma, and in patients who are abruptly discontinued from clonidine, propranolol will cause a rise in blood pressure in these situations. Exogenous administration of epinephrine to patients taking propranolol also will result in hypertension which can be severe. The hypertension occurs in these situations because of the unopposed alpha-adrenergic effect of epinephrine on the vasculature while the beta-adrenergic effects are blocked by propranolol. A similar effect would be seen with other beta-adrenergic blocking drugs such as nadolol that are not cardioselective, but the cardioselective drug metoprolol will probably not produce as much hypertension in these situations where circulating epinephrine occurs.

Vasodilators

Adverse drug interactions involving the peripheral vasodilators are rare. In general, volume expansion tends to counteract the effects of vasodilators and volume contraction tends to improve their effect on blood pressure. Also, many vasodilators cause activation of sympathetic nervous system and these effects can be blocked by sympatholytics. Therefore, the combined use of a sympatholytic and vasodilator with a diuretic is making use of advantageous drug interactions.

Interactions with Anti-Arrhythmic Drugs

Digitalis Glycosides

The most widely used cardiac glycoside in the United States is digoxin. This drug is not completely bioavailable when given in tablet form, and because of its relatively poor absorption, changes in gut motility or function can influence the amount of drug that is absorbed into the circulation.[8] Drugs that depress gastrointestinal motility may actually enhance bioavailability of digoxin probably by allowing the drug to maintain contact with the absorbing surface of the small bowel for a longer period of time. In a similar way, drugs that enhance gastrointestinal motility may decrease digoxin bioavailability. Some drugs may bind to digoxin in the bowel and alter its bioavailability. The most notable examples of this probably are the steroid binding resins such as cholestyramine which also can affect digitoxin absorption. Neomycin also has been shown to decrease digoxin absorption, perhaps by altering intestinal function.

The hepatic metabolism of digitoxin can be enhanced by inducers of hepatic microsomal enzymes. These drugs include phenobarbital, phenytoin, and phenylbutazone and results in a decrease in digitoxin blood level during any given dose used for chronic therapy.[1]

An interesting interaction between digoxin and quinidine has recently been reported. When quinidine is given to a patient receiving digoxin, the digoxin levels increase and digitalis toxicity may occur.[9-11] The mechanisms of this interaction appears to be two-fold. Quinidine can displace digoxin from tissue binding sites into the blood thereby acutely raising the blood concentrations. Quinidine also can decrease the clearance of digoxin and this results in a sustained increase in blood concentration of digoxin. Whenever quinidine is given to a patient that is also receiving digoxin (or probably also digitoxin) the dose of the cardiac glycoside should be decreased by 50% and blood levels monitored.

Pharmacodynamic interactions with the digitalis glycosides are fairly common as outlined above under Diuretics. Electrolyte changes produced by the diuretics can enhance digitalis toxicity even though the blood levels of the digitalis glycoside are not changed. Therefore, attention must be paid to the potassium, magnesium, and calcium concentrations of patients receiving diuretics that may affect digitalis effects. In addition, digitalis glycosides have effects on cardiac rhythm that can add to the effects of sympatholytic drugs. Thus, excessive sinus slowing or atrioventricular block can occur when digitalis is combined with a drug that also blocks the sympathetic nervous system. Sometimes this is advantageous, but other times it results in severe bradycardia that may compromise cardiac output.

Quinidine

Pharmacokinetic interactions involving quinidine include the quinidine-digoxin interaction mentioned above. Also, quinidine is largely metabolized by the liver mixed function oxidase system. Therefore, anything that could enhance the activity of this sytem, such as the enzyme inducers phenobarbital and phenytoin, will enhance the metabolism of quinidine.[12] This interaction can be clinically important if the enzyme inducer is either added to or subtracted from a regimen containing quinidine. On the one hand, the quinidine levels will fall, perhaps to subtherapeutic range, in patients where an enzyme inducer is added, and on the other hand quinidine levels may rise to the toxic range when an enzyme inducer is subtracted from such a regimen.

Pharmacodynamic interactions include the interaction of quinidine with the neuromuscular blocking effects of curare-type drugs frequently used during surgery. Cases have been reported where patients recovering from such drugs will have increased neuromuscular blockage upon the addition of quinidine. Quinidine can also, on occasion, increase the anticoagulant effect of warfarin.[13] The mechanism by which quinidine adds to the effect of warfarin is apparently at the vitamin K receptor rather than an effect on the pharmacokinetics of warfarin. Although this is not a universal effect, caution should be taken when quinidine is added to a patient who is stable on warfarin.

Disopyramide

Drug interactions with disopyramide seem to be of the pharmacodynamic type. Disopyramide has a similar effect to quinidine on warfarin efficacy.[14] When

disopyramide is added to a patient who is stable on warfarin, the anticoagulant effect of warfarin can be increased. The exact mechanism of this interaction is unknown. Another important pharmacodynamic interaction is the additive effect of disopyramide and other cardiac depressants on myocardial function.[15] Thus it has been reported that when disopyramide is given to patients receiving beta-adrenergic blocking drugs, severe and sometimes fatal cardiac depression and heart failure can occur. It is recommended that disopyramide not be used with other cardiac depressants because of the potential gravity of this drug interaction.

Lidocaine

Drug interactions with lidocaine are uncommon. It is known that the clearance of lidocaine from the body is largely dependent upon drug delivery to the liver, and diseases or drugs that decrease liver blood flow can decrease the clearance of lidocaine. Congestive heart failure will require that the doses of lidocaine be reduced. In experimental animals it has been shown that propranolol, which decreases cardiac output and liver blood flow, can also decrease the clearance of lidocaine.[16] The importance of this interaction in the clinical setting is unknown. In other experimental animal studies it has been shown the drugs that increase liver blood flow, such as isoproterenol, can enhance the removal of lidocaine from the body. Again, the clincial relevance of these observations is unknown.

Propranolol

The interactions with propranolol have been discussed under antihypertensive drugs.

Bretylium

The new antiarrhythmic drug bretylium has a guanethidine-like action in blocking the sympathetic nervous system. Bretylium enters the sympathetic neuron via the norepinephrine pump and, therefore, drugs that block the uptake mechanism for norepinephrine will block the sympatholytic effects of bretylium. This interaction has been used to advantage in an occasional patient who develops symptomatic orthostatic hypotension while receiving bretylium. Tricyclic antidepressants or ephedrine are effective in decreasing the orthostatic hyptension in such patients. It appears that the antiarrhythmic effects of bretylium are not blocked by these drugs, implying that the antiarrhythmic effects and the sympatholytic effects of bretylium are two distinct actions.

When bretylium is first given intravenously, catecholamine release occurs. Although usually of little consequence, the catecholamine release may worsen digitalis toxic arrhythmias. Certainly experimental animal models suggest that digitalis toxicity is a relative contraindication to the use of bretylium.[7]

Conclusion

Cardiovascular therapy with multiple agents is common. Most of the interactions between these drugs are useful and form the basis for multiple drug therapy. Occasionally, adverse interactions with antihypertensive and anit-arrhythmic drugs occur. As long as these interactions are anticipated, appropriate alterations in doses usually can be made. A most helpful laboratory test for pharmacokinetic interactions is the blood level of the drugs involved. If an interaction is expected, the blood levels should be monitored so that appropriate adjustment in doses can be made. For pharmacodynamic interactions, the blood levels will not be helpful and clincial observations must suffice for dosage adjustment. There are only a handful of drug combinations that should be avoided because of the potential for life-threatening adverse interactions. These include disopyramide with other cardiac depressants, bretylium and digitalis toxicity, guanethidine and drugs that block its entry into the sympathetic neuron, and monoamine oxidase inhibitors with drugs that can release norepinephrine from nerve endings. Other than these few interactions, the clinician can use combinations of drugs even though interactions are expected as long as he has well defined therapeutic goals so that altered amounts or effects of drugs will become evident. Drug interactions are a fact of modern cardiovascular therapy and should not be overly feared, but then again they should not be ignored.

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Seminars in Endocrinology and Metabolism

Hyponatremia

A Pathophysiologic Approach

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Introduction

Normal man regulates the tonicity of his extracellular fluid (ECF) volume with extraordinary precision, allowing constancy of the total body water over a wide range of fluid intake. This homeostasis is achieved through the function of the kidney's concentrating and diluting mechanisms, which are in turn dependent upon the appropriate release or suppression of vasopressin, a normal hemodynamic state, and normal intrinsic renal function. This discussion focuses upon hyponatremia, a situation in which with the very rare exception of extreme compulsive water ingestion, the diluting capability is impaired. In order to consider disorders of this mechanism, a brief review of normal water excretion is appropriate.

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The Normal Diluting Apparatus

Renal water excretion begins with the ultrafiltration of plasma at the level of the glomerulus. As the isotonic filtrate is reabsorbed isosmotically in the proximal nephron no solute-free water is generated there, thus any water appearing in the final urine is derived from more distal nephron sites. The next step in water excretion is dependent upon the function of the ascending loop of Henle. There sodium chloride is transported actively from the luminal fluid to the interstitium, with two important physiologic consequences. First, the extrusion of sodium chloride renders the remaining intraluminal fluid solute-free, thus generating free water and initiating the process of urinary dilution. Second, the accumulation of interstitial solute provides the driving force for the subsequent abstraction of water as the final urine courses through the terminal collecting duct. Thus, any factor interfering with the transport of sodium chloride in this nephron segment (e.g., furosemide and ethacrynic acid) impairs both the diluting and concentrating ability of the kidney.

As the urine reaches the cortical distal convoluted tubule, more sodium chloride is transported from the luminal milieu, resulting in progressively hypotonic urine until the entry into the cortical collecting duct. This sodium transport, and the further urinary dilution which results from it, is inhibitable by thiazides and their congeners. The dilute urine resulting therefrom must then pass through the cortical and medullary collecting duct, where the presence of antidiuretic hormone (ADH) will permit the reabsorption of water and the concentration of the final urine.

Pathophysiology

From the above analysis it is apparent that the ability to dilute the urine, and thus avoid the retention of excessive body water (and body solute dilution which is manifest as hyponatremia), hinges upon the integrity of the above mechanisms. That is, to dilute the urine normally, the individual must 1) maintain normalcy of the glomerular filtration rate (GRF); 2) reject filtrate from the proximal nephron, permitting delivery to the more distal diluting segments; 3) possess, in those distal segments, a normal capacity to form solute-free water; and 4) be able to suppress the release of ADH normally. Any process which interferes with any of these steps will, therefore, inhibit normal diluting ability and, depending upon the water intake, promote the development of hyponatremia.

A word on the quantitative aspects of the kidney's diluting capability is in order. The normal adult has a GFR of approximately 150 L/d. As approximately 20% of the filtrate may reach the distal diluting segment, with maximal suppression of ADH the normal individual can excrete up to 30 L/d. Thus, in the absence of an impairment of water excretion, a patient would have to ingest over 30 L/d of water to overwhelm the kidney's diluting capacity and cause hyponatremia. In the absence of such an extreme circumstance, which could only

occur if such a disturbed individual employed the assistance of a garden hose or shower, it is impossible for water ingestion alone to produce hyponatremia in normals. Thus, the occurrence of hyponatremia in association with lesser intakes of water is prima facie evidence that either a more potent stimulus than tonicity has led to the persistent release of ADH, or intrarenal factors have impaired the subject's capacity to dilute the urine and maximally excrete solute-free water.

A Clinical Approach

Hypontremia is a commonly encountered laboratory abnormality, which always should be explicable in the context of the above discussion. The subsequent consideration of the hyponatremic states will thus attempt to incorporate both a practical bedside approach and a pathophysiologic rationale.

First, a caveat about the diagnosis of hyponatremia. In some circumstances a decreased serum sodium concentration does not reflect a decrease in the osmolality of body fluids or a derangement in water balance. In cases of hyperglycemia the main cause of the hyponatremia is the glucose-related increase in osmolality in the ECF volume. This increase in osmolality is then associated with movement of water from the intracellular to extracellular compartment thereby resulting in hyponatremia. In order to correct for this phenomenon, we generally add 1.6 mEq/L of sodium to the measured sodium concentration for every 100 mg% glucose over 200 mg%. The "pseudohyponatremia" of hyperlipidemia and severe hyperproteinemia results from an artifact in the measurement of the sodium content of the laboratory specimen. Though the sodium concentration in the aqueous phase is normal, the sodium concentration in the total plasma may be diminished because of the volume occupied by the lipids or proteins. This pitfall can be circumvented by measuring the sodium concentration of the plasma water after the lipids or proteins are removed therefrom.

One more basic principle deserves emphasis. It is axiomatic that the serum sodium concentration describes nothing of the patient's total body sodium content, hence hyponatremia may exist with a low, normal, or elevated total body sodium. Our approach to hyponatremia relies heavily upon the determination of the patient's volume state, and upon an assessment of renal sodium handling as reflected in the measurement of urinary sodium.

With the current widespread availability of serum sodium determination has developed an appreciation of the spectrum of clinical abnormalities associated with hyponatremia. The severity of the clinical picture relates more directly to the rapidity of the fall in serum sodium than to the degree of hyponatremia per se. The manifestations range from no symptoms; to vague, nonspecific complaints including lassitude, anorexia, and leg cramps; to severe central nervous system manifestations including confusion, obtundation, and/or seizures. Regardless of the severity of the clinical manifestations the cause for the abnormality must be sought, which involves attention to several specific clinical data.

The Bedside Approach

First, the state of the patient's ECF volume must be ascertained. This requires a thorough history with attention to other concomitant medical diseases, any drug ingestion, and any other process (such as vomiting, diarrhea, or extraordinary cutaneous losses) which may influence the patient's volume state. The physical examination is helpful when evidence of fluid overload (edema, ascites, rales, ventricular gallop) or volume depletion (orthostatic tachycardia and hypotension, diminished skin turgor, absence of normal axillary and inguinal perspiration) is present. Though the determination of the volume state is of critical importance in approaching the hyponatremic patient, and though it is very easy at the extremes of volume overload and depletion, it is often very difficult to discern subtle deviations from the euvolemic state which are, nevertheless, of physiologic importance.

Routine Laboratory

The routine laboratory may be of help in determining the status of the ECF volume. While no direct measurement of the plasma volume is of practical clinical use, volume depletion may be associated with an increased hematocrit or plasma albumin concentration. A metabolic alkalosis may be observed if the ECF volume loss results from diuretics or vomiting, whereas a hyperchloremic acidosis would be a clue to prior diarrhea. In states of dehydration, the slow flow of glomerular filtrate through the nephron permits the increased reabsorption of filtered urea from the tubular fluid, whereas the filtered creatinine is not reabsorbable. Thus, the prerenal azotemia of volume depletion will often be associated with a disproportionately larger increase in the blood urea nitrogen than in the serum creatinine. While there are few clues to overhydration in the routine laboratory, it has recently been noted[1] that the mild, hypotonic volume expansion seen in the syndrome of inappropriate ADH (SIADH) may be associated with hypouricemia.

Once the patient's volume status has been assessed and the routine clinical and laboratory data has been gathered, a schema which classifies patients on the basis of their volume status and urine sodium excretion is useful, and is explicated below.[2-4]

Hyponatremia with an Expanded ECF Volume and Low Urine Sodium

In congestive heart failure (CHF), cirrhosis, and nephrotic syndrome the kidneys avidly retain sodium, contributing to the edema which characterizes these disorders. This antinatriuretic tendency usually manifests as a urine sodium, in a "spot" urine sample, of less than 10 mEq/L. Thus, the presence of hyponatremia

in an edematous patient with a low urine sodium implicates one of these disorders, which are usually readily discerned from the standard clinical examination.

Congestive Heart Failure

Hyponatremia in CHF is very common, through mechanisms which are not yet completely elucidated. The major hypotheses proposed to explain the diluting defect are 1) persistent secretion of ADH in response to nonosmolar stimuli (i.e., the abnormal hemodynamics of CHF), and 2) impaired distal delivery of glomerular filtrate to diluting sites due to increased proximal tubular reabsorption coupled with a decreased GFR. Though clinical and laboratory evidence for both can be marshalled, our bias, supported by our recent observations that detectable ADH is present in over three-fourths of hyponatremic patients with CHF,[5] is that persistent, nonosmolar stimulation of ADH release is of primary importance. However, central and intrarenal mechanisms are not mutually exclusive, and an interplay of both abnormalities is quite plausible.

Cirrhosis and Portal Hypertension

Systemic and renal hemodynamics are altered in cirrhosis, thus hyponatremia may be explained by the same basic mechanisms invoked in CHF. As in CHF, evidence in man and animals can be found to implicate both persistent secretion of ADH, as well as impaired distal delivery due to abnormal intrarenal hemodynamics. Recent studies from our institution[6] have underscored the role of ADH, as Brattleboro rats with congenital diabetes insipidus who developed cirrhosis nevertheless retained a capacity to excrete water which was indistinguishable from noncirrhotic Brattleboro rats.

Nephrotic Syndrome

The pathogenesis of the hyponatremia sometimes seen with nephrotic syndrome may be difficult to delineate. In those patients with diminished renal function the decreased GFR per se may contribute to the diluting defect. In some patients, with hypoalbuminemia and edema, it is likely that a decreased effective and absolute circulating blood volume results in a baroreceptor-mediated stimulation of ADH release. Impaired distal fluid delivery may not occur if the hypoalbuminemia diminishes proximal tubular reabsorption by decreasing peritubular capillary oncotic pressure.

Hyponatremia with an Expanded ECF Volume and High Urine Sodium

Acute and Chronic Renal Failure

The profound decrease in GFR seen in both acute and chronic renal failure impairs the ability of the kidney to excrete water. Recall that normally the daily GFR is approximately 150 liters, with a distal delivery (and hence potential free water excretion) of 30 L/d. In renal failure, however, a GFR of 5 ml/min or 7 L/d results in the distal delivery of roughly 2 L/d. Therefore, even if the patient can dilute the urine relative to plasma, the free water clearance is severely limited by the small amount of filtrate reaching the distal diluting site. Water intake in excess of the kidney's decreased ability to excrete water will thus result in hyponatremia. In both acute and chronic renal failure there may exist fluid overload and hence edema. Nevertheless, the tubular capacity to reabsorb sodium is impaired, thus the urine sodium is higher than 20 mEq/L.

Hyponatremia with a Diminished ECF Volume

By definition, the hypovolemic patient has to have sustained the loss of ECF. The routes by which the salt and water deficit may occur may be viewed as renal and extrarenal, with the urine sodium being high (>20 mEq/L and low (<10 mEq/L) respectively. The presence of hyponatremia, hypovolemia, and either a high or low urinary sodium excretion should suggest the following differential diagnosis.

Hyponatremia with Hypovolemia: Renal Losses
- High Urine Sodium -

Diuretic Excess

All diuretics, by promoting ECF volume depletion, may impair water excretion by decreasing distal fluid delivery and stimulating ADH release. In addition, the loop diuretics (ethacrynic acid and furosemide) and thiazides impair the renal diluting capability by interfering directly with free water formation in the medullary and cortical diluting segments, respectively. The additional contribution of diuretic-induced hypokalemia to this diluting defect remains speculative. It is postulated that potassium deficiency promotes a sodium for potassium shift across the cell membranes, or that hypokalemia somehow stimulates the release of ADH.[7] Although neither of these mechanisms is proven, it does appear that the correction of the potassium deficiency may correct the hyponatremia.

Addison's Disease

The mechanism of the diluting defect in Addison's Disease has been debated extensively.[8] Much of the controversy centers about the relative importance of mineralocorticoid and glucocorticoid deficiency in contributing to this abnormality. While it is clear, on the basis of studies in our laboratory[9,10] and elsewhere,[11] that glucocorticoid deficiency induces the nonosmolar stimulation of ADH release, it is also apparent that mineralocorticoid deficiency may contribute independently to the impairment of water excretion. The defect related to mineralocorticoid deficiency is mediated through volume depletion, which in turn stimulates ADH release, and alters renal hemodynamics. Thus, states of combined hormonal deficiency are associated with renal sodium losses, volume depletion, the nonosmolar release of ADH, and an impaired GFR, all of which participate in the observed diluting defect.

Salt-Losing Nephropathy

True salt wasting in renal disease is relatively rare, seen mostly in medullary cystic disease. However, all patients with chronic renal failure have a sluggish or

incomplete adapation to a low sodium intake. Thus, an abrupt decrease in sodium intake, or extrarenal sodium losses may lead to ECF volume contraction and cause hyponatremia. Patients with bicarbonaturia, whether from proximal renal tubular acidosis or during the initial bicarbonaturia after vomiting, may also develop hyponatremia on the basis of the renal sodium losses obligated by the bicarbonate present in the urine.

Hyponatremia with Hypovolemia: Extrarenal Losses - Low Urine Sodium -

In all the following entities the kidney responds normally to the depletion of the ECF volume, and demonstrates an appropriate antinatriuresis.

Diarrhea and Vomiting

Prolonged diarrhea and vomiting may induce severe, even life-threatening, contraction of ECF volume. Ingestion of water without sodium under these circumstances may lead to hyponatremia. It is felt that the renal diluting mechanism is impaired by both the nonosmosmolar stimulation of ADH release due to volume contraction, and an increased proximal reabsorption which reduces distal delivery.

Severe Burns and "Third Space" Problems

Patients with severe burns transude considerable fluid and protein through the damaged skin. Patients with "third space" fluid problems such as intestinal obstruction, acute pancreatitis or peritonitis, or extensive muscle trauma or necrosis may sequester large quantities of ECF in the damaged tissues. In all these situations ECF volume contraction ensues, and hyponatremia may result from both intrarenal alterations and the ADH release evoked by hypovolemia.

Hyponatremia with Normal or Modestly Expanded ECF Volume

For all intents and purposes, the hyponatremic patient who appears euvolemic on clinical examination will be either myxedematous, taking drugs which impair water excretion, demonstrating the physiologic release of ADH in response to physical and/or emotional stress, or will qualify for the diagnosis of the syndrome of inapproprate ADH (SIADH) secretion. The latter is not a disease, but rather the final common pathophysiologic pathway of a number of different disease processes. In addition, the relatively uncommon entity of isolated glucocorticoid deficiency as seen in hypopituitarism is discussed.

Myxedema

The mechanism underlying the hyponatremia of hypothyroidism remains to be established.[2] A lively debate has centered upon the relative importance of inappropriate secretion of ADH versus intrarenal alterations resulting in the decreased formation of free water. Others have postulated that relative glucocorticoid deficiency may play a role. Finally, the observation that active sodium transport depends upon normal thyroid function has engendered the speculation that the intracellular accumulation of sodium, coupled with a defect of water transport between extracellular and intracellular fluid volume, may be contributory to the hyponatremia seen in myxedematous patients.

Drug-Induced Defects in Water Excretion

The list of drugs implicated in impairing water excretion is extensive and growing,[12] and includes such agents as vincristine, cyclophosphamide, carbamazepine, clofibrate, chlorpropamide, and many others. The mechanisms by which these agents impair free water clearance involve either the nonosmolar release of ADH, or the potentiation of its hydroosmotic effect at the level of the renal tubule, or both.

Stress States

Physical pain and states of anxiety are known to be associated with the nonosmolar release of ADH. This stress response is pertinent to many patients who are postoperative or who have pain and anxiety for a variety of other reasons. An extreme expression of psychic stress is the psychotic state, in which euvolemic hyponatremia has been well documented and is felt to result from nonosmolar ADH release.

Isolated Glucocorticoid Deficiency

Hypopituitarism and consequent glucocorticoid deficiency are known to impair water excretion. The mechanism of this defect has been debated, with considerable controversy revolving about the importance of ADH versus an intrinsic renal abnormality. The diluting defect can be explained by two mechanisms, which are not mutually exclusive. It is felt, on the basis of considerable experimental data, the glucocorticoid deficiency promotes the baroreceptor-mediated, nonosmolar release of ADH, as discussed earlier.[8] Alternatively, it has been suggested that the impermeability of the distal nephron is incomplete in the absence of glucocorticoid hormones despite maximal suppression of ADH. Though studies in our laboratory[9,10] and elsewhere[11] support the primacy of vasopressin in this water-retention, evidence for both proposed mechanisms exists, including our observation that late in the course of glucocorticoid deficiency the diluting defect is ADH-independent.[10]

SIADH

The SIADH is discussed last because its diagnosis hinges upon the exclusion of every other cause of hyponatremia discussed heretofore. The diagnosis of the SIADH also requires a less than maximally dilute urine in the presence of a low plasma osmolality. The urine sodium is greater than 20 mEq/L when the patient is euvolemic and on a normal salt intake, but can be diminished appropriately in the face of volume-depletion or a low sodium intake.

A detailed exposition of the causes of the SIADH is beyond the purpose of this paper. However, the long list of known causes is greatly simplified by recalling that virtually any manner of intracranial pathology, many pulmonary diseases including infections and tumors, and a number of extrapulmonary tumors head the broad categories which require consideration. The occurrence of the SIADH without a known source is well

described,[13] and dictates constant vigilance for an identifiable cause. Of note is the recent attention focused upon patients with underlying chronic diseases who are said to have a "reset osmostat."[14] These patients are persistently hyponatremic but are able to dilute their urine below 100 mOsm/kg H_2O, and can excrete 80% of an administered standard water load in four hours. Whether this phenomenon truly implicates a basic alteration in the osmoreceptor, or whether it simply indicates that greater than normal reductions of serum osmolality are required to inhibit potent nonosmotic stimuli remains disputable.

Therapy of Hyponatremia

Therapy of the hyponatremic state is based on the correct interpretation of the physiopathologic mechanism causing the disorder. Ideally, the underlying conditions causing the impaired water excretion should be corrected whenever possible. For example, treating congestive heart failure, removing diuretics or other pharmacologic agents, administering thyroid hormone in myxedema or steroid hormones in Addison's disease, and repletion of ECF volume with isotonic saline in cases of gastrointestinal losses or salt losing nephropathy will correct the hyponatremia. Water restriction is the mainstay of therapy in SIADH unless more definitive measures are possible, i.e., surgery or chemotherapy in cases of SIADH associated with tumors.

An enhancement of solute-free water excretion is a logical way to deal with hyponatremia if sodium depletion and ECF volume are not present. In this regard, administration of demeclocycline in cirrhosis and in patients suffering from SIADH in an effort to diminish the responsiveness of the collecting duct to ADH has been attempted.[15,16] However, in cirrhotic patients, this drug seems contraindicated due to a reversible renal toxicity manifested by a decrease in inulin and para-aminohippurate clearances.[15] However, with SIADH some success has been reported with this approach.[16] Lithium carbonate also may produce nephrogenic diabetes insipidus but this occurs in only 10% of patients and thus is not consistently effective in treating SIADH.[17] Schrier et al[18] used furosemide in seven patients suffering from hyponatremia in edematous disorders including CHF, cirrhosis, and nephrotic syndrome. In five of these patients, the diuretic induced excretion of dilute urine, an effect not abolished by the simultaneous administration of ADH. The mechanism involved may be rapid flow of urine in the collecting duct which impairs complete osmotic equilibration with the medulla, or perhaps inhibition of the action of ADH.

Sometimes, especially in SIADH, profound acute hyponatremia demands emergency correction of the lowered serum sodium concentration. The traditional approach to this problem has been administration of hypertonic sodium chloride. This approach is fraught with two disadvantages: 1) the correction of hyponatremia is usually transient because the patient rapidly excretes almost all of the administered sodium, and 2) the patients with SIADH are already volume-expanded and thus administration of hypertonic saline can lead to circulatory overload especially in elderly patients. Hantman et al[19] circumvented this problem by administering furosemide to these patients and replacing their urinary electrolyte losses with hypertonic (3%) sodium chloride and appropriate amounts of potassium chloride. This led to a negative free water balance without volume overload. In five patients suffering from nontumoral SIADH, hyponatremia was corrected in a matter of hours without complications. This form of therapy thus is indicated when SIADH is accompanied by life-threatening hyponatremia. In more chronic forms of SIADH, water restriction still remains the treatment of choice, in addition to more definitive treatment.

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Seminars in Gastroenterology and Liver Disease

Esophageal Dysphagia

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Dysphagia is one of the most important and underestimated symptoms in medicine. In general terms, dysphagia means "trouble with swallowing." For the purposes of this paper, only esophageal dysphagia will be discussed and oropharyngeal swallowing problems will be reserved for a subsequent discussion.

When a patient complains of food sticking somewhere between the throat and the stomach there is an extremely high incidence of anatomic or neuromuscular abnormality. For practical purposes, dysphagia is almost never a hysterical symptom. Although emotional upset may aggravate an underlying defect, dysphagia must be carefully evaluated and not be attributed only to "nerves."

When evaluating patients with dysphagia, a detailed clincial history is extremely important. Listed below are a number of historical features to be evaluated:

1. **Esophageal obstruction:** The consistency of food that sticks is very helpful in distinguishing true atomic obstruction from neuromuscular disease. Patients with a narrow esophagus have more difficulty with solids than liquids. They frequently have difficulty with food such as bread or meat. Patients who have esophageal spasm or achalasia may have equal difficulty with liquids or solids.

2. **Duration of symptoms:** A prolonged duration of symptoms is most consistent with a benign disorder. Rapidly progressive dysphagia is suggestive of neoplasm. A history of intermittent esophageal obstruction is characteristic of an esophageal Schatzki "B" ring.*

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*The Schatzki "B" ring is a web-like ring which occurs at the esophageal gastric mucosal junction. This ring may be intermittent and only seen with full distention of the esophagus or may be a persistent ring and actually represent a true stricture.

3. **Localization of symptoms:** When patients are asked to localize the site of obstruction, they are very accurate if they point somewhere between the sternal notch and the xyphoid. If they point at the sternal notch itself, localization of symptoms is not as accurate. When patients are asked to localize the position of a barium-coated marshmallow at fluoroscopy, they invariably point to the marshmallow.

4. **Symptoms of gastroesophageal reflux:** Heartburn and regurgitation commonly, but not always, accompanies peptic "B" ring strictures. Interestingly, as obstructive symptoms increase, reflux symptoms may improve due to the esophageal protection offered by the stricture. The physician may have to inquire into the past for symptoms of significant reflux esophagitis. When patients with strictures subsequently undergo esophageal dilation, the reflux symptoms frequently recur.

5. **Odynaphagia (painful swallowing):** Odynophagia is most often seen in inflammatory processes involving the esophagus such as Monilia or viral esophagitis or in patients with esophageal ulcer (Barrett's esophagus). Occasionally odynophagia may be a manifestation of tumor invasion. An unappreciated fact is that symptoms of odynophagia due to Monilia esophagitis may be seen even in low dose antibiotic use (Tetracycline or Erythromycin) commonly prescribed for acne. This mild Monilial infection involves the mid esophagus and is easily treated by temporary elimination of the drug and Mycostatin mouth wash (swish and swallow 500,000 units, 4 times a day).

6. **Esophageal Colic:** This is a severe pain, presumably due to esophageal spasm, which may radiate across the precordium and into the back. This pain can be confused with coronary pain due to the distribution and severity

as well as the frequent response of esophageal pain to Nitroglycerin.

Although this pain probably does represent spasm of the esophagus, it should not necessarily be attributed to the condition, idiopathic diffuse esophageal spasm. Most esophageal spasm is secondary to inflammation, obstruction or neoplasm and these entities should be excluded radiographically and endoscopically. A demonstration of diffuse esophageal spasm on esophageal motility studies does not establish idiopathic diffuse esophageal spasm unless these other entities are excluded by x-ray and endoscopy.

7. Recurrent pulmonary infections: Recurrent bronchitis and chronic obstructive pulmonary disease may occur as a result of aspiration of gastric contents. This clinical sequence is often difficult to prove. Patients with an obvious history of awakening frequently at night aspirating bitter material represent the minority of patients who suffer from recurrent aspiration.

8. Gastrointestinal bleeding: Gastrointestinal bleeding either acute or chronic should be searched for in all patients who suffer from dysphagia.

Clinical Evauation of Patients with Dysphagia

1. Upper G.I. Series: The upper G.I. series remains the initial screening study for patients with dysphagia. It is important, however, to realize that a normal esophagram does not exclude a significant lesion. On the other hand, the presence of a hiatus hernia is not enough to explain the patient's symptoms of dysphagia and careful evaluation searching for true stricture of neuromuscular abnormality must continue. In ordering an upper G.I. series it is important to alert the radiologists as to the complaint of dysphagia. The radiologist should then pay careful attention to proper distention of the esophagus with barium. In patients with esophageal stricture, either benign or malignant, it is unusual to experience dysphagia unless the diameter of the stricture is less the 13mm. This critical diameter should be kept in mind when evaluating the upper G.I. series in patients with swallowing difficulty.

2. Upper G.I. Endoscopy: This is probably the most important diagnostic test and has an extremely high yield particularly in patients who have a history of true bolus obstruction. A full upper G.I. endoscopy should be performed. In addition to evaluating the esophagus, careful examination of the cardia should be performed to exclude a gastric carcinoma in this region. The gastric outlet is examined carefully to exclude obstruction as another aggravating factor in gastroesophageal reflux.

3. Esophageal Motility Study: Esophageal motility studies should not be performed prior to endoscopy since the presence of the motor disturbances characteristic of the above lesions does not eliminate the need for endoscopic evaluation.

4. Esophageal motility is important in the diagnosis of Achalasia and other specific disorders which may be associated with esophageal spasm. Motility studies are useful in establishing esophageal spasm if the study is strongly positive. However, minor abnormalities in motility tracings are common and do not establish a diagnosis. On the other hand, patients with severe spasm may have a normal tracing since spasm is frequently an intermittent disorder.

Common Causes of Esophageal Dysphagia

1. Peptic stricture
2. Esophageal "B" ring
3. Esophageal ulcer (Barrett's esophagus)
4. Carcinoma
 a. squamous
 b. adenocarcinoma (gastric)
5. Achalasia
6. Scleroderma
7. Diffuse esophageal spasm (idiopathic)
8. Monilia esophagitis
9. Viral esophagitis

5. Bernstein Test (acid profusion test): This test is most useful in the differential diagnosis of chest pain, i.e., coronary pain vs esophageal spasm. It is of little value in the evaluation of dysphagia unless combined with an esophageal motility study. Acid profusion during an esophageal motility study will sometimes produce manometric abnormalities which is particularly useful information if the patient's clincial symptoms are elicited at the same time.

6. Cine-esophagram: The cine-esophagram is rarely useful in evaluation of esophageal dysphagia but frequently quite valuable in the workup of patients with oropharyngeal dysphagia.

Management of patients with dysphagia is, of course, dependent on etiology. It is beyond the scope of this paper to discuss management of all types of dysphagia. I would like to discuss however, the management of patients with esophageal stricture.

The majority of patients with esophageal obstructive symptoms have either peptic stricture or it's variant, the esophageal "B" ring. These patients can usually be managed quite well with a combination of anti-reflux measures and esophageal bougienage. The goal of bougienage is to dilate the esophagus above the critical diameter of the esophagus where most patients develop dysphagia. (13mm or approximately 39 French). Patients are dilated using mercury filled bougies to 46-50 French and dilated periodically thereafter, depending on the severity of the process leading to the stricture formation.

Esophageal stricture should not, in itself, be taken as an indication for anti-reflux surgery. Some patients may, however, desire surgery because of the return of significant reflux symptoms after dilatation or because of the need of frequent bougienage.

In summary, patients with dysphagia should always be evaluated carefully. An extremely high yield of significant, treatable problems can be anticipated.

Nutrition

The Nutritional Approach to Intrauterine Growth

Retardation - Fetal Malnutrition

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Fetal malnutrition (FM) is a chief cause of prepartum death in utero.[1] It is associated with considerable high mortality and morbidity during labor, delivery and the neonatal period.[2] and significantly impairs growth and development of children.[3,4] FM is a public health problem relative to the number of children affected and the socio-economic consequences of their decreased potential for productivity.

The wealth of information accumulated over the past 20 years indicates that FM can be regarded as a condition susceptible to treatment.[5] It requires the cooperative efforts of physicians, nutritionists and other health care providers; unfortunately these are sometimes separated by traditional practices that parted the care of the mother from the care of the child.

There is increasing evidence that at least two types of intrauterine growth retardation can be recognized with ultrasonic biometry.[6,7] These two types are manifestations of impaired growth in different stages of intrauterine development; one type shows evident growth retardation relative to gestation at all stages of pregnancy, and is probably the result of a reduced number of cells. The other type of FM is more common, and occurs in the later months of pregnancy with evident signs of soft tissue wasting and characteristically reduced Rohrer index ($100 \times {}^{W}/L^{3}$) or ponderal index, with decreased fat deposits as measured by skinfold thickness.[8] From the clinical, epidemiological and nutritional points of view these two types of growth retardation are separate entities, although as parts of the continuous dynamic process of intrauterine growth, it may be difficult to separate them in practice.

Considerations regarding the etiology and prevention of diseases such as rubella toxoplasmosis and cytomegalo-virus infections or congenital malformation, chromosomal and otherwise, implicated in the first trimester growth retardation are beyond the scope of this paper; albeit important as a health problem, constitute only a small proportion of the infants affected by intrauterine growth retardation.[9]

Diagnosis

Fetal malnutrition of the third trimester constitutes a major challenge to the diagnostic capabilities of even the most modern obstetric services.

Multiple pregnancy is unquestionably a significant risk factor for fetal malnutrition; at the University of Arizona Health Sciences Center, twins usually plot one

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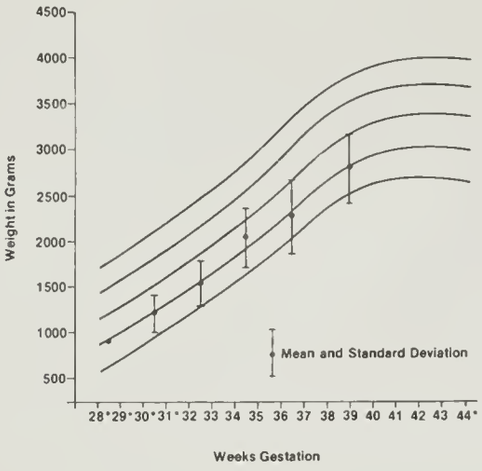


Figure 1
Weight of twins grouped by 2-week gestational age; mean and standard deviation. *projected lines.

standard deviation below the mean weight for gestation (Figure 1). To compound their problems the majority of them are born prematurely, indicating that diagnosis and special care are needed in the early stages of pregnancy when it is still possible to provide therapeutic intervention.[10] Ultrasonic examination at mid pregnancy enjoys a good reputation as a safe and reliable method of diagnosis in multiple pregnancy, and is the only reliable method to assess growth in utero by serial measures.[11] It is not known to what extent early and adequate nutritional intervention plus controlled activity could reduce the incidence of FM and prematurity in multiple pregnancy. Preliminary data seem to indicate that there are possible benefits from such an approach, but the undesirable effects have not been evaluated.

Maternal malnutrition has been implicated as one of the most common problems leading to FM world wide. Maternal pre-pregnant weight significantly affects the incidence of FM,[12] as does the total number of Kilocalories consumed during pregnancy.[13]

Epidemiologic, clinical and field studies have completely changed our perspective on nutritional aspects of pregnancy. It has been shown repeatedly that mothers who have had one infant with FM are likely to give birth to another FM, that maternal pre-pregnancy weight but not height influences the weight of the infant at birth, and that supplementation of calories during pregnancy is associated with a comparable increased birth weight.[14,15]

In the Western world, sub-optimal protein and caloric nutrition during pregnancy seems to be inevitably tied to socio-economic status with a gradient of quality food intake directly related to economic status.[16]

What constitutes an adequate diet during pregnancy is a subject of heated debate. For the time being, the United Nations Food and Agriculture Organization (FAO/WHO) recommendations seem sensible: 0.52 gm of quality protein (milk or egg variety) per kg per day, plus 1 extra gm at the beginning of pregnancy, increasing to 9 gm by the last trimester.[17] Calorie recommendations are more difficult to establish since it is so definitely related to the activity of the individual pregnant woman; besides, differences in methodology renders counts of calories difficult to standardize. 2200 K. cal. per day for a moderately active 55 Kg woman could be used as base line, with additional 80,000 K. cal during the entire pregnancy.[17]

How should the health care provider determine that the patient is taking an adequate diet in daily practice? Dietary history, to be reliable, requires such tedious and detailed count, that it would be unrealistic to expect reliable accounts from the patients.[18] The obvious, albeit less exact, way to assess energy intake is weight and fat thickness measures.[19]

Weight gain during pregnancy of 12 Kg has been set by several organizations as the ideal,[19] although allowances are made for increased weight gain for the low pre-pregnancy weight woman, and some decrease to about 10Kg for the overweight. However, mention should be made that it is not recommended that a woman participate in weight-reduction diet during pregnancy.[20]

Cigarette smoking is probably the one drug habit which affects fetal growth the most, and is also the one that has received the most attention. Consistently every study of the effect of smoking in pregnancy has shown that there is an association with reduced birth weight, and that this effect is independent of socio-economic status, race, sex, and maternal pre-pregnancy weight.[21]

Regardless of the specific effect of cigarette smoking, the resulting increased incidence of low birth weight infants is a public health concern. Suggestions have been made that poor nutrition is the real problem leading to FM, and that dietary supplementation could reduce the impact of maternal smoking.[22] However, a study involving a very large population sample has shown that there is a direct effect of maternal smoking on fetal growth, possibly due to decreased availability of oxygen in the presence of carbon monoxide in the mother's blood, and that of the fetus as well.[23]

Alcohol use (and abuse) during pregnancy has received a great deal of attention in the medical and lay press in the past few years with the recognition of specific teratogenic effects of alcohol; the fetal alcohol syndrome, including reduced birth weight and mental retardation with characteristic facial features.[24] Although the full syndrome is related to heavy drinking, lesser amounts of alcohol may cause an increased incidence of FM.[25]

Other causes of FM, the epidemiology of which group has an impact on public health are: (a) dietary fads, (b) drug use and abuse, (c) diseases such as diabetes groups D, E. and F, (d) reno-vascular disease, and (e) toxemia of pregnancy (toxemia gravidica), perhaps the most important in terms of frequency. Recent studies would implicate this disorder beyond question,[1] and its effects have been linked not only to growth retardation but also to adverse effects upon the central nervous system.[26]

In summary, it should be emphasized that adequate perinatal nutrition can play a role in the reduction of FM, and that attention to nutritional factors in pregnancy may bring an improvement in perinatal mortality and morbidity which will be of significance to overall public health. A plea is made to diagnose FM in utero (and multiple pregnancy) early enough to provide comprehensive nutritional and medical care designed to increase weight in utero and to decrease prematurity and intra partum complications in this group of children.

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Oncology

Cancer Therapy:
An Emerging Role for Physical Medicine and Rehabilitation

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Abstract

There are many complex problems which arise in the management of the cancer patient as the disease progresses from one stage to another. The specialist in physicial medicine and rehabilitation joining with his medical and surgical colleagues can contribute his special skills to both the diagnosis and management of this disease and its treatment related complications.

The multifacted team approach blending the skills of the physiatrist, therapists and social worker as successfully employed in the rehabilitation of chronic neuromuscular disability can with equal facility be utilized in the management of the cancer patient.

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Although the rehabilitation challenges of hemiplegia, amputation, para and quadraplegia, neuropathy and myopathy remain essentially the same, treatment time is telescoped by progressive disease, requiring frequent alterations in the "staging" of functional levels of locomotion and self-care.

The physiatrist, resourceful in meeting the physical and social requirements of living with a severe disability must become at least in part able to cope with the needs of those also dying.

In December of 1971, former President Richard Nixon signed a bill authorizing the expenditure during the next three years of $1.6 billion for cancer care and research. This act focused the nation's attention on neoplastic diseases as a major cause of death in our country, second only to renal and cardiovascular disease. A projected absolute increase in yearly mortality due to exceed 400,000 deaths by the year 2000 accompanied by increasing morbidity as patients live longer responding to sophisticated treatment, provides a growing challenge to all branches of medical specialization. Increasingly, this challenge is being met by the combined talents of individual specialists working in a coordinated manner. At William Beaumont Hospital, the Division of Physical Medicine and Rehabilitation plays an integral role in this response -both diagnostically and therapeutically. We have often been both the first specialty contact for the patient and with increasing frequency, the last.

The few articles dealing with the rehabilitation of the cancer patient have pictured this activity based either at a specialty cancer hospital or rehabilitation institute.[1,2] Our practice, conducted at the community level is based at a 940-bed, acute-general-care hosptial, where busy services admit over 30,0000 patients a year. Formal arrangements are not immediately available for extended or rehabilitative bed care; however, individually and as a team, the various specialty resources of the Physical Medicine and Rehabilitation unit are available to the 'acute' patient both for in- and outpatient therapy.

The multifacted team approach blending the skills of physiatrists, therapists and social worker as successfully employed in the rehabilitation of chronic neuromuscular disability have been with equal facility utilized in the management of the cancer patient.

Although the rehabilitative goals of hemiplegia, para- and quadriplegia, amputation therapy, neuropathy and myopathy may remain essentially the same, treatment time is often telescoped by progressive disease requiring frequent alterations in the "staging" of the functional levels of locomotion and self-care. The direct or remote effects of the cancer itself, the residuums of radical surgery and the toxic side effects of chemo- and radiotherapy, combined with a sympathetic understanding of the 'dying process' individually or together are the challenges the physiatrist must master to successfully join the oncology 'team.'

Cerebral metastasis is the most common neurologica complication of systemic carcinoma.[3] Lung and breast are the most frequent sources with the large bowel pancreas and renal carcinoma also important primaries Approximately 20 to 40 percent of tumors are metastatic to the brain with 10 percent of cases having multiple site of metastasis. In more than half the cases, patients with carcinoma of the lung and malignant melanoma develop brain metastasis.

Primary tumors of the central nervous system account for 2 to 5 percent of all tumors with 80 percent involving the brain. Gliomas account for 50 percent of all primary brain tumors and of these, glioblastomas are most frequent. Among the non-gliomatous tumors meningiomata are most prevalent with pituitary adenomas, primarily chromophobic, representing 12 to 18 percent of intracranial neoplasms.

In children most primary tumors are located in the posterior fossa. Medulloblastomas and astrocytomas are each present in 30 percent of cases with ependymomas found only in 12 percent.

Approximately 40 percent of these patients presenting with hemiplegia secondary to brain metastasis can be restored to a useful and productive life with an additional 30 percent gaining palliation. Early identification, surgical decompression, corticosteroids in massive doses and external irradiation together or separately can temporarily produce a restoration of function. The symptoms of hemiparesis and aphasia can clear dramatically when compared to the more frequent slower resolution of symptoms following a vascular occulsive episode.

The 4,000 new annual cases of primary spinal cord tumor are comparatively rare. Meningiomas and Schwannomas occurring most frequently at thoracic level account for 56 percent of the total. Gliomas, primarily of the ependymoma type, represent another 23 percent of the total with half arising in the filum terminale or conus medullaris. A miscellaneous group less frequently found are the epidermoid and dermoid cysts, hemangioblastomas and chondromas.

Extradural metastasis with spinal cord compression is an all too frequent complication of systemic cancer Autopsy studies have domonstrated epidural metastasis in over five percent of all cancer patients. The most common primary sources are breast, lung, Hodgkin's disease and prostatic carcinoma. Ninety percent of these patients initially present with pain localized to the site of spinous metastasis. Too often, weeks to months pass before a radicular pattern of pain and weakness develop sufficient to demand intensive diagnostic study. In 50 percent of patients with metastatic disease initial 'negative' x-ray and nuclear scan examinations can delay appropriate treatment. In these early stages electromyographic examination can be useful in suggesting the presence of metastatic disease when the neurological, nuclear scan and x-ray examinations are

otherwise normal. Earlier identification and agressive treatment of spinous metastasis can yield a greater precentage of independent, ambulant patients.

Electrodiagnostic studies are also helpful adjuncts in localizing sites of direct metastatic compromise of peripheral nerves as well as identifying the confusing assortment of neurological syndromes associated with the remote effect of carcinoma.

Carcinoma of breast and prostate are common sources of cauda equina compression. Carcinomas of the bladder, cervix, and prostate can all be locally invasive producing lumbo sacral plexus dysfunction. Local invasion of the brachial plexus by superior sulcus tumors can present with medium cord compression Horner's syndrome and ipsilateral recurrent laryengeal nerve paresis. Lymph node enlargement secondary to lymphoma or metastatic carcinoma as well as direct metastasis to bone are all capable of also producing localized pressure neuropathy.

The syndromes frequently associated with carcinomas and often present preceeding its identification are those of neuromyopathy, polymyositis and the myasthenic syndromes. Each separately and/or as a complication of central nervous system metastasis can be identified by electrodiagnostic studies.

A rehabilitation program essentially unchanged from the multidisciplined team approach utilized in the management of chronic neuromuscular disability due to vascular or traumatic insult is employed in treating the cancer patient. No singular recipe is available which, like a magic encompassing formula, can be utilized to treat all patients with a similar clinical presentation. Instead, the rehabilitation program must be tailored to the individual's disabilities. A sophisticated rehabilitation program is a dynamic process, pragmatic in its approach to the disability, it relies on a series of daily structured 'tests' to identify residual abilities as a substitute for lost function.

Rehabilitation goals can be predicted with greater certainty when they are established with regard to the natural history of the primary cancer, the therapeutic medical-surgical approach and its complications, as well as the patient's current clinical status.

The prevention and management of contractures, bowel and bladder dysfunction, decubitus and mental depression is prerequisite to the establishment of an active neuromuscualr rehabilitation program.

The initial response of the patient to this program is one of the primary determinants in assessing tolerance for more stressful physical activity. Pain due to remote metastasis, fatigue associated with a limited cardiopulmonary reserve and weakness related to the presence of neuromyopathy, among other symptoms, are additional factors to be considered when activity levels are established. An initial excellent response productive of an ambulent patient, independent in self-care, can be frustrated by progressive disease or systemic response to the toxic side effects of therapeutic treatment.

The rehabilitation team effort through a series of re-evaluations can return the cancer patient to an active and productive life. Unfortunately, with equal frequency the evaluations can culminate in program changes to accommodate increasing disability: inital efforts directed at ambulent self-care may be reoriented to wheelchair and assisted self-care activity and finally to supportive, total bed care. Early in the evaluation, efforts must be made to mobolize family and community resources to provide assistive and supportive home care if discharge planning is to be expedited. Family involvement, necessary at all levels of function, is imperative during the final stages of supportive care. Equipment including hospital beds, wheelchairs, lifts and other nursing items can be obtained on loan through the 'cancer closet' of the local unit of the American Cancer Society. Nursing assistance provided by the Visiting Nurse Association (VNA) is an invaluable adjunct to patient home management. Although instructions in patient bed care, including ostomy and catheter management, are provided to the family prior to discharge, continued supervision by the visiting nurse is essential.

The clinical presentation of the cancer patient is an ever changing one demanding close and attentive follow-up. Clinical 'labels' of hemiparesis or paraparesis are not always sufficient to explain new symptoms of pain or weakness. Pain during assistive joint range of motion may be the first indicator of a new metastasis or pathological fracture; progressive weakness the initial sign of an increasing pressure myelopathy may require emergent decompressive surgery.

The rehabilitation team can, with an active, positive and sophisticated approach to therapy, help to overcome the sense of dispair and helplessness which accompanies the diagnosis of disseminated cancer. Less fearful of death itself then overwhelmed by the "process of dying" with its attendant anxieties associated with decreased pain tolerance, increasing dependency and fear of abandonment, some patients completely withdraw refusing all therapeutic overtures. Most, however, never losing hope welcome any and all emotional and physical support. A continuing major effort must be made by the rehabilitation team to bridge the gulf between those who treat and cannot cure and those who will otherwise die abandoned to grief and loneliness.[5,6]

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Pediatrics

Sex Chromosome Abnormalities and Noonan Syndrome

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Abstract

Among the most frequently encountered genetic disorders are the sex chromosomal abnormalities. Their presentation is subtle in the early pediatric period, usually being more prominently manifested in the pubertal child. A general review of sex chromosomal abnormalities is presented with emphasis on incidence, clinical signs, and significant problems of Turner, Klinefelter, and XYY syndromes. Noonan syndrome, which is frequently confused with Turner syndrome is also discussed and clinical signs are contrasted.

Sex chromosome abnormalities are among the most frequently encountered genetic disorders. Yet, because of their subtle manifestations or late, post-pubertal presentation, they are often missed. The purpose of this paper is to review the common sex chromosome abnormalities: Turner syndrome, Klinefelter syndrome and the XYY syndrome, as well as some general principles of the ways in which these sex chromosome aneuploidies may occur. Finally, there will also be a short discussion of Noonan syndrome, which is not a chromosome disorder but has many clinical similarities to Turner syndrome.

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For many years the chromosome number in man was thought to be 48. It remained for Tjio and Levan in 1956 to demonstrate that the correct number was 46. The 46 chromosomes include 44 autosomes and 2 sex chromosomes. Today, chromosome pairs are numbered consecutively from 1 to 22 according to decreasing chromosome length and centromere position. The twenty-third pair, or sex chromosomes, are out of order. They form an identical pair—XX—in the female, but are markedly dissimilar in the male—XY.

What is so remarkable about the sex chromosomes is that their abnormalities create more business for the physician than all the other 44 chromosomes in Man.[1] This comes about neither because the sex chromosomes are more prone to anomalies than the autosomes, nor because the disorders are more severe. Rather, sex chromosome abnormalities seem to be less damaging. The individual affected by a sex chromosome anomaly tends to survive, and is often less affected by the underlying disorder. A majority of sex chromosome abnormalities are not noted until puberty. Autosomal abnormalities frequently eventuate in a spontaneous abortion or lead to death within the first year after birth.

Forty-six chromosomes are not essential for human survival. Children may have extra chromosomes. However, there is only one type of monosomy (lack of a whole chromosome) that is consistent with life, this involves the X chromosome and leads to Turner syndrome. YO embryos never survive. Autosomal monosmy is likewise lethal.

How do sex chromosome abnormalities arise? During meiosis accidents may occur resulting in nondisjunction, or improper segregation of chromosomes—this may occur during the first and/or second meiotic division—leading to gametes with an excess or deficient chromosomes complement. The various combinations of these gametes are depicted in Table 1.

Other chromosomal accidents may occur which involve the division of the chromosome during meiosis such that an isochromosome is formed. This happens when the centromere splits in such a way that a chromosome has the same arm on both sides of the centromere and so has a duplication of one arm and loss of the other arm. The resultant chromosome is genetically inactive. Other abnormalities of chromosome structure, such as deletions and duplications, are well known.

Sex chromosome mosaicism may occur with two or more different sex chromosome complements. Because of mosaicism and also subtle morphologic chromosome changes, it should be obvious that when a sex chromosome abnormality is clinically suspected, chromosome studies rather than buccal smears must be done to characterize the sex chromosome complement.

Some general points may be noted concerning the clincial characteristics of the major sex chromosome abnormalities. First, abnormal stature is one of the few consistent traits associated with these disorders. XO

Table 1
Sperm

		X	Y	XY	O	XX	YY
	X	XX Normal	XY Normal	XXY Kline-felter	XO Turner	XXX Triplo-X	XYY
O V A	O	XO Turner	YO Lethal	XY Normal	OO Lethal	XX Normal	YY Lethal
	XX	XXX Triplo-X	XXY Kline-felter	XXXY Kline-felter	XX Normal	XXXX Tetra-X	XXYY Kline-felter

individuals are unusually short. An increased number of sex chromosomes (particularly the Y) makes for increased stature. Second, mental capacity may be normal, but tends to be skewed toward the low normal to subnormal level in individuals with an abnormal sex chromosome constitution.[2] Third, fertility is generally decreased.

Turner Syndrome

In 1938 Turner described a series of post-pubertal females who exhibited short stature, webbed neck, cubitus valgus, and failure to enter adolescence.[3] Albright in 1942 demonstrated that these patients had elevated urinary gonadotropins.[4] Wilkins and Fleishman in 1944 described the "streak gonads" associated with the syndrome. Ford[5] first described the XO karyotype in 1959.

Turner syndrome occurs with an incidence of 0.4/1000 live female births.[6] The usual sex chromosomes constitution in Turner syndrome is XO. About 80% of Turner patients are XO. The remaining 20% have structural anomalies of the X such as isochromosomes or are sex chromosome mosaics.

There is a high fetal wastage in Turner syndrome. In excess of 95% of all Turner conceptuses are spontaneously aborted.[7]

The origin of the X chromosome in Turner syndrome has been studied using X-linked traits such as color-blindness and the Xg blood group. The solitary normal X chromosome in Turner syndrome is maternal in origin in about 70% of cases, i.e., the chromosome error in Turner syndrome is paternal in at least 70% of cases. There is no maternal or paternal age effect in cases of Turner syndrome.

Girls with Turner syndrome are short. As adults they generally range from 120-147 cm in height, with a mean of 140 cm. The only clinical clue to the diagnosis of Turner syndrome prior to adolescence may be short stature. Growth retardation is a continuum in this syndrome. Small size is often evident at birth. The mean birth weight is 2900 grams. Height and weight increase after birth at a slower rate than normal. There is also a striking lack of adolescent growth spurt. One should suspect Turner syndrome in any girl two standard deviations below the mean height for her age.

Features of Turner syndrome are evident in the fetus. The webbed neck develops from lymph-filled sacs at the nape of the neck. Webbing of the neck is present in approximately 50% of the newborns with Turner syndrome.

The neck is usually short with a low posterior hairline. The hair at the nape of the neck may extend to the shoulder level. The anterior hairline is also often low.

Lymphedema of the dorsum of the hands and/or feet may be present at birth. Turner syndrome should be considered if an infant girl has puffy hands or feet. The finger and toenails in Turner syndrome tend to be hypoplastic and hyperconvex.

About 70% of girls with Turner syndrome exhibit cubitus valgus, an increased carrying angle of the arm. There is often a "shield chest" with widely spaced, hypoplastic nipples.

Radiographically, the most revealing signs are shortened fourth metacarpals and drum-sticking of the distal phalanges. However, Bercu and associates[8] have suggested that the reticular, osteoporotic pattern of the carpal bones even in prepubertal subjects is a more consistent sign than those previously mentioned.

About 20% of patients with Turner syndrome have congenital cardiovascular defects, most often coarctation of the aorta (70%). A significant number of the remainder have aortic valve disease or atrial septal defects.

Systemic hypertension in the absence of coarctation is not an infrequent finding and should prompt search for other etiologies, notably renal abnormalities. The problem with hypertension is oftentimes a significant one. Careful consideration should be given before routine prescription of estrogens as part of therapy, as will be discussed later.

Renal malformations are common in Turner syndrome.[9] Estimates range widely from 15-90%. Typical anomalies include horseshoe kidney, ectopic and bifid kidneys, malrotation, and multicystic kidney. Some workers[10] advocate intravenous pyelograms for all patients with Turner syndrome.

Hearing loss is much increased in incidence in Turner patients.[11] For unknown reasons, otitis media occurs frequently. It is not surprising then that there is an increased incidence of conductive hearing loss. There is also an increased incidence of nerve deafness as well. The auditory problems of the patient with Turner syndrome are often not appreciated. All girls with Turner syndrome should have audiologic examinations.

At the time of puberty, patients with Turner syndrome present with sexual infantilism. The nipples are small and hypopigmented. Breast tissue is absent. The vagina and external genitalia appear normal. The uterus is small. The ovaries are rudimentary fibrous

streaks. There are usually no oocytes present, and when there are, it is probably due to mosaicism. There are a few reported cases[12,13] in the literature of XO Turner syndrome patients who were able to reproduce, but these patients were probably mosaics with a normal XX cell line.

Even in patients with mosaicism, fetal wastage is high.[38] One-half of pregnancies are aborted, and one-fourth have congenital anomalies including a remarkably high incidence of Trisomy 21, congenital heart disease and hydrocephalus. The risk of a Turner child to a Turner mother is only 0.4%. As one can appreciate, the chance of having a normal child is especially low, and patients should be advised early.

Approximately 10% of Turner patients have diabetes and there is a high incidence of hypothyroidism.[11,14] This is especially true with Turner mosaicism where one study[15] indicated a 50% occurence of thyroid disease.

If there is a mosaic line containing a Y chromosome as in XO/XY, the incidence of gonadoblastoma is considerable and gonadectomy should be performed.

Psychologically, it is important to realize that Turner syndrome is not an intersex problem. Patients are unquestionably female. Efforts should be made to provide every opportunity for the Turner female to appear as normal as possible—this can be approached from the aspects of stature and secondary sexual development.

Current therapeutic recommendations advocate initiation of androgens to increase long bone length at about age ten years. Androgenic therapy may be prescribed before feminization with estrogens, and is most effective in the first 12-18 months of therapy. Halotestin 2.5-5.0 mg/day or Oxandrolone 0.075-.125 mg/kg/day may be employed.[16] Height, bone age, and virilizing side effects should be followed every three to six months until the bone age is about thirteen years (when the distal thumb epiphyses fuse). Androgens are then discontinued and estrogen—Premarin 0.625-1.25 mg/day is initiated until break-through bleeding begins. Cyclic estrogen therapy is then begun. Provera, 5 mg/day on days 14-21 may be added if breast development is inadequate.[17]

Following this general protocol, one can maximize growth at a time which is psychologically important to any teenager and also initiate secondary sexual development which is equally important. One must follow all precautions usually taken in patients taking estrogens.

Klinefelter Syndrome

In 1942 Klinefelter and his associates described a series of postpubertal males having small, firm testes with tubular hyalinization, azospermia, gynecomastia, increased urinary gonadotropins and decreased urinary 17-ketosteroids.[18] In 1956 Plunkett and Barr[19] described a similar series of males with positive nuclear chromatin bodies. With the advent of karyotyping, Jacobs and Strong[20] in 1959 first published data on XXY karyotypes in chromatin positive males.

Klinefelter syndrome, hereafter used to designate any male karyotype with at least two X chromosomes and at least one Y, occurs with a frequency of 0.9/1000 live male births.[21] Klinefelter syndrome is probably the most common sex chromosome disorder. Eighty per cent of patients have XXY, 10% are XY/XXY mosaics, the remaining 10% are of diverse karyotypes.

Maternal age is significantly increased in XXY Klinefelter syndrome with a mean maternal age of 32 years. About 60% of XXY Klinefelter males have only maternal X's as shown by the segregation of X-linked traits.[6,22]

The somatic features of Klinefelter syndrome usually do not become apparent until puberty. Prior to puberty, body proportions are near normal. At puberty the body takes on a eunuchoid habitus being slightly taller than normal, with long lower extremities and long arm span (3 cm greater than height). The testicles fail to enlarge and remain small and firm, usually less than 2 cm in length. Microscopically, the seminiferous tubules appear hyalinized with very little germinal epithelium. The Leydig cells are clumped in groups.

The penis is normal in size. The testes are usually descended. The prostrate is small. Facial hair is sparse. Most patients either do not shave or shave infrequently. Axillary hair is also sparse and pubic hair distribution may resemble the female escutcheon. Libido and potency may be decreased. There have been very few cases of fertility. Klinefelter syndrome is often discovered during male infertility work-ups, totaling about 10% of males investigated.[23]

In about three-quarters of cases, intelligence is normal with about a quarter of patients being mildly retarded. This is an important point to consider in a male child with learning problems. This may be the only clue to early diagnosis.

Significant gynecomastia develops in about 50% of patients. There is a sixty-fold greater incidence of breast cancer in Klinefelter syndrome as compared to normal males.

Patients with higher numbers of X chromosomes have an increasing degree of mental retardation, as well as skeletal anomalies such as radioulnar synostosis. This minor skeletal anomaly is also a good clue to a prepubertal diagnosis of Klinefelter syndrome.

Opinions on treatment modalities in Klinefleter syndrome vary.[24] Adequate studies have not been done to establish the safety and efficacy of systemic androgen therapy. The only satisfactory method of management for significant gynecomastia is mastectomy.

XYY Syndrome

Sandberg and coworkers[25] first reported a man with an XYY karyotype in 1961. Interest in the XYY syndrome was aroused in 1968 when Casey[26] and Jacobs[27] reported a disproportionately high incidence (2-4%) of XYY indivudals in prisons and mental hospitals.

The incidence of XYY is approximately 1/1000 live male births. XYY arises from paternal nondisjunction

during the second division of meiosis.[21] Since 1-2% of all sperm contain two Y chromosomes, it would seem obvious that there is some selection against YY sperm.

Clinically, the XYY male after age 5-6 is usually over the ninetieth percential for age in height.[6] Both leg and trunk length are increased, but the leg/trunk ratio is normal. The facial features may be assymetric. There may be mild pectus excavatum and scapular winging.

Sexual development is usually normal. XYY males are usually fertile. Their offspring are usually chomosomally normal although XYY and Trisomy 21 offspring have been documented.[28]

Extensive psychiatric evaluation has been carried out and published concerning XYY prisoners. In general they have been noted to have explosive behavior and decreased preception of consequences.[29,30] They have shown a propensity to destroy property rather than to attack individuals.

With an estimated occurence of 1/1000 live males, there is a large number of these individuals in our society. If all of these were violent criminals, our prisons would be filled with XYY individuals. The XYY syndrome appears at a tenfold increased frequency in criminals, but it would be unwise to consider the syndrome as an automatic predisposer to violent criminality.

Noonan Syndrome

Noonan syndrome is not a sex chromosome abnormality. However, because of similar facies, it is often confused with Turner syndrome.

In 1930 Ullrich[31] described an eight-year-old girl with webbed neck short stature, cubitus valgus, congenital lymphedema, ptosis, and small nipples. There was no allusion to sexual infatilism, because of the child's age. A remarkably similar phenotype with sexual infantilism was described by Turner[3] as previously noted in 1938. In 1949 Ullrich [32] again published, recounting his 20 years of observations and noted a preponderance of females in a 4:1 ratio.

In 1963 Noonan and Ehmke[33] reported on the increased incidence of congenital heart disease found in these patients. Polani[34] in 1969 called attention to the normal sex chromosome constitution and normal secondary sexual characteristics. Nora and coworkers[35] in 1972 documented an apparent autosomal dominant mode of inheritance in certain families.

The clinical characteristics of Noonan syndrome are somewhat similar to Turner syndrome. Short stature is the rule. Eighty-three percent are significantly shorter than their first-degree relatives.

The characteristic Noonan facies include narrow maxilla and small mandible. Hypertelorism with slight antimongoloid slant to the eyes is common. The ears are minimally low-set and sometimes posteriorly rotated. Neck webbing occurs in 50%, associated with a low posterior hairline.

Congenital heart disease occurs in 35-50% of cases with predominance of right-side cardiac lesions. In Noonan syndrome, pulmonic valvular stenosis is most common, but this is often associated with an ASD. Asymmetric septal hypertrophy has also been reported, as well as pulmonic branch stenosis.[36]

Table 2
Comparison of Turner & Noonan Syndromes

Chromosomes	XO, mosaic, isochromosome, etc.	Normal
Sex	Female	Female to male 4:1
Incidence	0.4/1000	0.4-1/1000
Stature	Short	Short
Facies	Similar	Similar
Heart Disease	Coartation, Aortic stenosis	Pulmonic stenosis
Renal Anomalies	Frequent	Rare
Sexual Characteristics	Infantile	Normal, but may have delayed puberty
Gonads	Streak ovaries	Usualy normal ovaries Crytorchid, infertile males

Common skeletal anomalies in Noonan syndrome include scoliosis, pectus excavatum, and shield chest.

There is usually normal secondary sexual characteristics but the onset of puberty may be delayed. Females are usually fertile; ovarian dysgenesis has been reported. There is a high degree of cryptrochidism with associated infertility in male patients.

Renal anomalies are rare. Mental capacity varies widely. The IQ is general ten points less than unaffected sibs.

There is a paucity of information on incidence, but one study[37] estimated the incidence at 0.4-1.0/1000. There have been reported cases of direct transmission of this syndrome from both mother and father to child, and thus some[35] have proposed an autosomal dominant mode of inheritance.

Table 2 contrasts some of the important characteristics of Turner and Noonan syndromes. Turner syndrome is a sex chromosome aneuploidy, whereas Noonan syndrome has a normal chromosome constitution. There has been documented autosomal dominant inheritance patterns in some families, so it is likely that Noonan syndrome is due to a single autosomal gene.

Noonan patients may be either male or female, but most often the latter. The incidence of both Noonan and Turner syndromes is similar. Both syndromes exhibit short stature and similar facies. Heart disease is predominantly left-sided in Turner syndrome and right-sided in Noonan syndrome. Renal anomalies are prominant in Turner syndrome, not in Noonan syndrome.

Turner patients, unless mosaics, are infertile with streak ovaries. They also exhibit sexual infantilism.

Noonan syndrome patients may experience delayed puberty, but sexual characteristics are otherwise normal. Females are generally fertile, while males are often cryptorchid and sterile.

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Psychiatric Disorders

The Family in Chronic Illness

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Introduction

The family in today's society represents a beleaguered unit beset by many problems faced by our society as a whole. The family unit as traditionally defined includes mother, father and children. Extended families in the past have offered support systems which have included grandparents, aunts and uncles, etc. The family generally has provided an element of equilibrium to offset stresses in economic, vocational and social spheres encountered by all members of the greater world.

The almost fifty percent divorce rate of today tends to color intensively the make-up of families. With such a high divorce rate, approximately half of all families have more than one set of "parents" relating to children. The issue of divorce in second marriages approaches sixty percent. Therefore, repetition is a frequently encountered factor suggesting that experience is not "always the best teacher."

Against this backdrop we find, even in emotionally stable or "healthy" individuals, stresses which societal changes have brought about that tend to disrupt and disturb efforts at equilibrium.

Illness is a disruptive factor to ego function for individuals and their families or units, with which they live. For instance, work situations are severely disrupted by illness of a key person. Families whose chief breadwinner becomes ill, are severely disrupted. The role changes in men and women, in today's society, makes the illness of either parent have impacts far beyond those of traditional family models.

When illness is acute, the changes are unanticipated and temporary. Although the illness may persist for some period, a resolution of the crisis occurs with either a total recovery or expiration. Chronic illness, however, represents a long term change to which an individual and their family must adapt.

Elements involved in chronicity include: 1) remissions and exacerbations; 2) permanent alteration of life style; 3) change in organization of work; and 4) alterations in degree of responsibility attributed to each family member. When illness involves a parent, changes impinge on social and sexual relationships.

There are several individuals or institutions with whom the family comes into constant contact. These represent lines of defense organized in an effort to reestablish equilibrium within the family unit. Thus, individuals may seek help through extended family, if stable; or friends, if substantial. But, beyond this, the institutions include clergy, educational, social service and medical. In the latter sphere, the family physician is often the first line of support and defense. The patient is seen initially. Beyond the initial contact, however, it is necessary for family to be included in any therapeutic approach. Thus, if a patient develops a chronic illness, it is important for the family to have conscious awareness of what that illness is like; what its remissions and exacerbations portend; what general concepts are relevant to short, intermediate, and long-term illness. Within such a setting, it is important to convey clear information to the patient so that they are aware of the potential impact of the illness.

Disruptions in communication may lead to "use of the illness" by either patient or family. Education, thus, becomes a therapeutic tool to enhance clarity where confusion reigns, and to provide stability where uncertainty exists.

This paper will endeavor to examine the implications of various diagnostic entities for patients and their families. Efforts will be made to evaluate optional approaches to enforcing, reinforcing, revising, and creating adequate mechanisms with which both patients

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and families can cope more effectively in a chronic illness.

Chronicity and Patient Response

Chronic illness and its treatment represent particular types of stress. Their impact on patients is determined by such factors as psychological genetic exposure to illness, hereditary predisposition, sex and role definitions, as well as existing and pre-existing support systems.

All illness activates psychological responses. Regression is one such adaptive response. Healthy regression allows for participation in treatment. "Some patients tend to regress and emphasize the helpless aspects of their illness; they become childish and silly, and in general adopt a 'sick role.' This 'flight into illness' tends to anger both the health care professional and the family of the patient; thereby further aggravating the already tenuous emotional status of the patient."[1] Both illness and regression leave a patient more vulnerable than before. Conflict arises between independence and dependence. A genuine fear of loss of control relates to the inability to make the illness "go away". Both self-esteem and body-image are involved, with reactions of men and women differing in some aspects.

Serious illness requires a reorganization of the family following the initial response of denial, anxiety and resentment. "The reorganized family may function as well as, or perhaps better than, it did before the crisis, or in the process of reorganization family roles and rules may change significantly," resulting in serious emotional pain or impairment of other family members. The family's reorganization may affect the patient's illness and recovery.[2]

The family of a patient with cancer may insist on avoiding discussion of the illness with the patient, protesting that they are protecting the patient. The dynamic explanation of such behavior may lie in their own wish to avoid or deny the painful circumstance. Thus, they may limit the patient's effective discharge of emotions relative to their perception of the illness. An angry withdrawal may distort the seemingly good intentions leading to strains in current communications and guilt at later times.[3]

Case History I

A twenty-seven year old married man sought consultation because of guilt over pending termination of his marital relationship. The patient was the oldest of six children. He was delegated much responsibility from early childhood. His mother frequently left town with the next oldest child, a sister. The patient was enraged over his responsibilities, but failed to express them emotionally. After three years of mother's trips, the sister died of leukemia. The patient felt a heavy sense of guilt over his unexpressed rage. Mother had never told him about sister's illness. His guilt over this undiscussed illness was a deep-seated factor of major importance in his guilt over potential abandonment of his present day family.

Family Equilibrium in Illness

Olsen[2] refers to the "impact of serious illness on the family system" stating that "serious illness is a family affair." He notes that the physician who treats seriously ill patients should keep several concepts in mind. 1) The entire family must adjust to the illness. 2) The state of an illness, such as coronary artery disease, affects the state of the family's, as well as the patient's, existence. 3) Serious illness often precipitates crisis, with resultant pressures that require reorganization within the family. 4) These reorganizations may affect certain family members more than others. For example, he refers to teenagers and their acting out either in school, sexual areas or physical expressions.

The medical needs of the patient do not exclude the needs of the family. Though the family presents a united front in protecting itself from intruders, the physician is one of the outsiders who can enter and help reestablish stability. The family physician may: 1) include the family during the acute phase, with conferences to allow an expression of affect; 2) include the family in discussions of illness, particularly if it is chronic; 3) recognize changing attitudes including anger and resentment, as well as feelings of depression; 4) allow the family to be included, at the time of dismissal, in discussions of post-hospital management of illness; and, 5) note changes in family interactions which might necessitate referral to a mental health professional.

The treating physician should alert the hosptial staff to be aware of family dynamics. The staff, thereby, may facilitate family reorganization in an adjunctive manner.

Shubin[4] discussed the impact of cancer on wives noting the differences involved between the impact of a spouse's death from cancer or cardiovascular disease.

Scheidman[5] described chronicity as a forced learning factor. She noted changes that chronic illness produces in life style, employment, finance, and family roles. The ongoing nature of chronic illness necessitates adaptations at various levels. She also underscored the importance of psychosocial factors on the recovery of patients with chronic illness.

Bruhn[6] stated that chronic illness is the most common form of illness. Management must include psychological and social factors for patient and family. Role changes and task reallocation represent ongoing factors which are more difficult to accept because of their long-term nature. Chronic illness may also create new problems in various family members who are required to assume different responsibilities. He noted that there are "high rates of family breakdown" in such illnesses as diabetes, hemophilia, and epilepsy when family members are unwilling or unable to readapt. When identified patients have exacerbations, healthy members may become "sick" to gain attention.

A University of Michigan study[7] observed that there were "substantial social effects of rheumatoid arthritis that are perpetuated between parental and conjugal families." It went on to state that women with

rheumatoid arthritis have marriages resembling those of their parents; but there was "status stress" reflecting discrepancies, at several levels, between their own status and that of their husband's. These women often felt and expressed a great deal of angery aggression toward husbands, who in turn reciprocated. There also was a significant frequency of peptic ulcer in husbands of women with rheumatoid arthritis. On the contrary, men with rheumatoid arthritis were found to be low in conscious feelings of anger and aggression, with a "rather benign attitude" reflected by a low level of hostility in their marriages to "healthy women."

Persons who try to change long standing roles in the family disrupt the family organization. Often this individual is labeled a "mental patient." It becomes important to define family roles, to understand the stage on which issues are acted out.

Krupp[8] noted that adaptation to chronic illness is "making the best of a bad bargain." He feels that the physicians can help only if the patient meets the challenge of adaptation. Often the patient can adapt, but the family is unable. Denial or non-acceptance may produce great strains. He stated also, that the "helping people" around the patient may exacerbate the challenge of compromise through unwitting criticism of the patient's difficulties with adaptation.

Therapeutic Concepts

The approach to treatment of chronic illness must focus initially upon the patient. When immediate medical needs have been met and acute crisis stablized, the treating physician and his supporting team should incorporate an effort to aid the patient's family in coping. Many centers today have begun didactic sessions with patients and families in an effort to clarify history and course of illness, as well as treatment. The focus of these sessions runs the gamut from medications to descriptions of remissions and exacerbations; elaboration of the meaning of illness; the impact upon physical relationships, including sexual; and, the impact of social stress upon family circumstances. Other areas include nutrition, physical therapy and ancillary therapies which are part of the treatment regimen. The Phoenix Arthritis Center is one such treatment center wherein rheumatologists, nurses, dieticians, physical therapists, and a mental health cotherapy team have joined teaching efforts crossing various disciplines. The value of such sessions is the relatively low anxiety level on which they operate. The meetings may lead to referals for depth-oriented therapeutic approaches in the psychiatric and psychological sphere. Because this program relates to rheumatologic disorders, it has unique qualities relevant to the illness. Parallel programs have been utlilzed in myasthenia gravis, diabetes, hemophilia, etc.

Where such sessions are not possible, it would be incumbent upon the physician to periodically incorporate the family throughout the patient's treatment. Mental health consultations may ensue.

Case History II

This family was seen because of agitation, sensitivity and tearfulness of a twelve year old daughter with juvenile rheumatoid arthritis of four years duration. The family included a ten year old son, mother and father who were in their early forties. Father had an executive position in industry, while mother had settled into a domestic role.

The patient was seen by the male therapist for six interviews prior to school summer recess. Mother was seen by the female therapist. Issues of guilt were noted in mother and daughter. Illness became a source of anxiety leading to a symbiotic interaction. Mother would overprotect and the patient would over-react. It was apparent that neither had their needs sufficiently met. Mother has worked on letting go of daughter and assuming more responsibility for her own growth, including school. Initially the patient was distraught over mother's new attitude. In therapy, she reviewed her concepts of the family. She began to accept mother's needs and acknowledged that she too could grow into womanhood with many options. The authors chose to treat this family individually because of the notable problems which the symbiotic relationship presented.

The use of exploratory psychotherapy is dependent upon the physician's perception of the patient's or family's needs. Udelman and Udelman[9] have worked with groups of rheumatoid arthritic patients in both inpatient and outpatient settings, finding that groups provide a good vehicle for sharing experiences in illness.

Summary

Chronic illness presents patients, families and physicians with ongoing challenges. These range from financial concerns, through treatment options to psychological support. Each member of the above triad requires continuing education. The understanding of illness and enforced adaptation is primary for the patient, and an awareness of present and future changes is helpful for the family. A global assessment of pathology, physiology and emotions is a primary requirement for the treating physician.

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Radiology

Case of the Month No. 43

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Cary J. Stegman, M.D.

A 63 year old white man came to emergency room complaining of right upper quadrant pain. An abdominal ultrasound examination was performed (Figure 1)

What organ is being imaged?

What causes the "shadowing" of the echos (small arrows)?

What is the diagnosis?



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Acute Renal Failure

Clinical Update

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Introduction

Acute renal failure (ARF) is defined as the sudden cessation of kidney function, with resultant retention of nitrogenous wastes and other solutes, which is not reversible with the manipulation of extrarenal factors. It should be noted that, while previous textbooks included oliguria in the diagnostic criteria, it is now widely appreciated that functional renal failure may occur despite the preservation of a normal urine output. This phenomenon is understood by recalling that then the renal concentrating capacity is normal, the maximum achievable urine osmolality may be 1200 mOsm/L. Normally, then, the normal daily endogenous solute load (approximately 600 mOsm) can be excreted in a urine volume of 500 ml. However, when ARF supervenes the maximum achievable urine osmolality may be only 300 mOsm/d, obligating a urine volume of two liters per day to maintain solute balance. Any urine volume less than that will result in solute retention and azotemia.

Pathogenesis

Though our understanding of the pathogenesis of this entity is incomplete, it is appropriate to review briefly the pathogenetic mechanisms postulated to contribute to the development of ARF. A detailed consideration of the experimental data for and against each proposed mechanism is beyond the scope of this discussion, but has been summarized elsewhere.[1]

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The observation that ARF may commonly follow ischemic episodes initially drew attention to hemodynamic events in the reduction of the glomerular filtration rate (GFR).[2] Whether such hemodynamic alteration would result from afferent arteriolar vasoconstriction sufficient to reduce glomerular capillary hydrostatic pressure, or activation of the renin-angiotension system has not been completely resolved. Alternatively, arteriolar endothelial cell swelling has been offered as a cause of diminished glomerular filtration.

Virtually all the nephron segments are included in some proposed pathophysiologic sequence. It has been suggested that the decrease in GFR is due either to an intrinsic decrease in the hydraulic permeability of the glomerular capillary wall, or that filtration is opposed by the increased intratubular hydrostatic pressure resulting from luminal obstruction. The latter has been observed in animal models,[3] and is thought to result from sludging of cellular debris. Others, however, have argued that glomerular filtration in fact proceeds normally, but that "backleak" of the markers used to measure GFR (e.g., inulin) across damaged tubular epithelia precludes meaningful data on GFR in this setting.

Although all of these factors may, at some time, contribute to the establishment of ARF, none of them alone seems to explain the severely reduced GFR. Our bias, based partially on animal studies in our laboratory, is that the development of ARF often incorporates more than one pathogenetic mechanism. For example, we favor the importance of hemodynamic events early, with a resultant decrease in glomerular capillary pressure. Later, secondary tubular obstruction may perpetuate the functional loss. In any case, a successful clinical approach to ARF, with an appropriate emphasis on prevention, requires some conception, however imperfect, of its pathogenesis

Clinical Setting

Historically, ARF has been associated with all varieties of surgical, obstetrical, and medical insult, ranging from obvious calamities such as shock states and crush injuries to more subtle insults such as seemingly uncomplicated general anesthesia and surgery. Over the past decades there has been an increasing awareness of several subgroups of patients with ARF, namely patients with nontraumatic rhabdomyolysis and with nephrotoxic renal injury. In view of their recently established etilogic prominence, these entities are discussed briefly below.

Rhabdomyolysis

Rhabdomyolysis, long known to occur in crush injuries, burns and inflammatory states of muscle, also occurs in a variety of settings in which muscle blood flow, oxygen supply or metabolism is disturbed or muscle energy consumption is increased. Two recent series[4,5] have emphasized the occurrance of nontraumatic rhabdomyolysis secondary to myopathies,

seizures, strenous exercise, or coma from drug overdose. It now appears clear that rhabdolmyolysis in this latter setting results from limb compression with consequent muscle ischemia.[6] Symptoms of muscle pain and weakness, and muscle swelling and tenderness were observed in roughly one half of the patients. The diagnosis was supported by a constellation of laboratory findings including orthotolidin-positive urine dipstick of RBC-free supernatant, pigmented granular casts, and elevated muscle enzymes. In addition, other characteristics abnormalities included rapid rises in serum creatinine, potassium, uric acid, and phosphate. In some patients, early hypocalcemia may be followed by late hypercalcemia during the diuretic phase. The latter abnormality probably results from the dissolution of dystrophic muscle calcification, and may lead to severe metastatic calcification.

Aminoglycosides

The aminoglycoside antibiotics have formed the cornerstone of therapy in gram negative bacillary infection for the past two decades. With their use, however, has developed a sizable incidence of iatrogenic renal disease. Though it is difficult to quantify the nephrotoxicity of a drug per se in patients whose illness and whose other drugs may cause or potentiate the development of ARF, it is well established that the aminoglycosides are, in themselves, nephrotoxic.

The spectrum of aminoglycoside-induced renal injury has been reviewed recently.[7,8] Early in the course of toxicity tubular proteinuria, enzymuria, aminoaciduria, renal glycosuria, and a concentrating defect may be demonstrated. With more pronounced nephrotoxicity the urinalysis shows proteinuria and cylinduria, and azotemia with an elevated creatinine may ensue. The reduced GFR may result in part from a decline in the glomerular capillary ultrafiltration coefficient.

The pharmacokinetics of aminoglycoside nephrotoxity have been investigated extensively in animals. It appears that the degree of injury parallels the extent of renal cortical tissue binding of the drug. A correlation between toxic potential, extent of renal tissue binding, and the number of free amino groups has been suggested, with the primary locus of drug uptake being the proximal tubule. Aminoglycosides can be concentrated up to twenty-fold in the renal cortex, thus tissue levels persist far longer than the serum half-life of the drug. This probably explains the observation that ARF from aminoglycosides can occur after the drug has been discontinued, and why recent exposure to aminoglycosides is a risk factor in predicting nephrotoxicity with drug administration. Other factors predisposing to aminoglycoside nephrotoxicity include advancing age (perhaps due to administration of a dose based upon an overestimation of the GFR); volume depletion; preexisting renal dysfunction; and prior administration of other nephrotoxins. Included in the latter category are anesthetic agents, radiocontrast materials, and furosemide, perhaps because of the diuretic's potent volume-depleting effect.

Of considerable clinical interest are two recent prospective, doubleblind, randomized studies of aminoglycoside nephrotoxicity from Johns Hopkins University. In the first, the combination of cephalothin plus an aminoglycoside was compared to methicillin plus an aminoglycoside. The former combination was found to result in an increased incidence of nephrotoxicity, suggesting that the interaction of a cephalosporin and an aminoglycoside promotes synergistic nephrotoxicity.[9] The second study compared the nephrotoxicity of gentamicin with that of tobramycin. The investigators found that in treatment groups with similar age, pretreatment renal function, and courses of antimicrobial therapy that tobramycin caused a lower incidence of nephrotoxicity that gentamicin.[10]

Regardless of which particular aminoglycoside is chosen, several precautions seem reasonable in an attempt to minimize or avert nephrotoxocity. First, the patient's extracellular fluid volume should be expanded. Second, the dose of antibiotic should be adjusted to the creatinine clearance, particularly in the elderly in whom renal function is often overestimated. It is prudent to avoid concomitant therapy with cephalosporins and other nephtotoxins if possible, and caution should be employed after prior courses of aminoglycoside therapy. It is axiomatic that these drugs should not be used unnecessarily, and that their empiric use should be discontinued as soon as is permitted by the clinical picture, results of cultures and sensitivities. The utility of monitoring plasma aminoglycoside levels has not been firmly established. While the concensus experience has found such levels useful in gauging therapy, as the drugs are handled by renal filtration alone, there is a theoretic basis for the argument that plasma antibiotic levels yield no more information than the serum creatinine. The resolution of this question awaits further clinical study.

Radiocontrast Materials

Our ever increasing reliance upon diagnostic radiocontrast procedures has resulted in many reports of radiocontrast-induced ARF. The overall incidence of the entity is variable between series, but appears to be less than one percent of all patients examined. However, the incidence may be under-reported, and is clearly higher in several high-risk patient groups, including patients with diabetes mellitus, the elderly, patients with prior renal insufficiency, and probably patients with multiple myeloma. Though dehydration appears to compound the existing risks in susceptible patients, ample evidence indicates that adequate hydration does not prevent postcontrast ARF. Hypertension, hyperuricemia, and dose of contrast media have been proposed, but not proven, to be independent risk factors. In contrast to aminoglycoside nephrotoxicity, ARF from contrast materials is most often oliguric; but like aminoglycoside-induced ARF, contrast-induced ARF, though usually reversible, may lead to irreversible renal impairment, the need for dialysis, and even death.

Diagnosis

The diagnosis of ARF hinges on the exclusion of pre-renal and post-renal causes of azotemia. Volume depletion and cardiac dysfunction are, by far, the most commonly encountered causes of pre-renal azotemia. Aortic dissection, renal arterial emboli, and peripheral vasodilatation should also be considered in a compatible clinical setting. The former diagnoses are more seriously entertained if renal blood flow is absent, which can be ascertained readily with isotopic renography.

Urinary tract obstruction should be sought in every patient with declining renal function. In our experience[11] obstruction accounts for roughly ten percent of the azotemia for which our service was consulted. Our suspicion should be particularly heightened in patients with a normal urinalysis, and in patients with a urine output which is totally absent, very variable, or unusually copious. The initial search for obstruction involves such fundamental procedures as a post-void residual catheterization to exclude bladder neck or urethral obstruction, a careful pelvic examination in all women to exclude an obstructing gynecologic process, and an abdominal flat plate to detect the ninety percent of renal calculi that are radioopaque.

The further approach to exclude urinary tract obstruction has undergone revision in the past decade. As the hazards of antegrade and retrograde urography have become more evident, so has grown the experience, sensitivity, and availability of ultrasonography. The ultrasongraphic diagnosis of urinary obstruction depends on sonolucency separating the normal central calyceal echoes. In a recent prospective evaluation[12] the sensitivity of ultrasound in excluding infection was found to be ninety-eight percent.

In the diagnosis of ARF much may be learned from the urinalysis and urinary electrolyte composition. Characteristically, the urinalysis reveals an active sediment with renal tubular epithelial cells, cellular debris, and tubular cell casts. A normal sediment suggests pre-renal or post-renal azotemia. The urinary electrolytes reflect the associated tubular dysfunction, with a "spot" urine, in the absence of prior diuretic therapy or preexisting renal insufficiency, showing a urine sodium over 20 nEq/L, and an elevated urine to plasma urea nitrogen and creatinine level. The derived index obtained by dividing the urine sodium over the urine to plasma creatinine (The Renal Failure Index) has been found to discriminate well between patients, with values less than one suggesting pre-renal azotemia or acute glomerulonephritis, and values greater than one implicating intrarenal ARF or obstruction.[11]

Course

Though ARF clinically has been associated with oliguria, our recent data discloses that more patients with ARF are nonoliguria than are oliguric.[13] The etiologic classification of the patients revealed a prominence of toxin-induced ARF in nonoliguric patients. The course of the patients differed markedly, in that the nonoliguric patients had a significantly lower mortality and morbidity than the oliguric patients, a finding in agreement with several other series. Of note was the finding that our patients who became nonoliguric after early furosemide therapy had a course indistinguishable from the other nonoliguric patients. This raises the question about the value of furosemide administration in an attempt to convert an oliguric to a nonoliguric state, a practice which, though potentially of value, has never been validated by prospective, controlled data. While this critically important question awaits further study, it is clear from the clinical and experimental data to date that, if furosemide administration is of value in attenuating the course of ARF, it will have to be given early in the course of oliguria. Additionally, extreme care should be employed to assure that all correctable pre-renal factors, especially volume depletion, are optimized before the administration of "prophylactic" furosemide.

Treatment

The management of ARF can be considered in terms of general, supportive care, and dialytic therapy. As infection remains the leading course of death in ARF, constant vigilance in the maintenance of intravenous lines, attention to pulmonary hygiene, and an avoidance of indwelling bladder catheters is mandatory. The early diagnosis and appropriate treatment of wound and other infections is urgent. Similarly, awareness of gastrointestinal bleeding as a serious cause of mortality in these critically ill patients dictates careful surveillance of the patient's vital signs, hematocrit, and tests for occult blood in the stool. The role of cimetidine in preventing gastric hemorrhage in this setting remains to be explored.

The intake of salt and water should be tailored to whether the patient is oliguric or nonoliguric. Serum electrolytes, BUN and creatinine, fluid intake and output, and weight must be carefully monitored. The serum phosphate should be controlled with appropriate antacids, and concurrently given drugs must be adjusted for the level of renal failure.[14-16] Adequate caloric and protein intake must be ensured in order to decrease wasting and improve general clinical status.

The latter prescriptions about protein intake reflect changing attitudes about dialytic therapy. Whereas dialysis was once received for overt uremia, refractory hyperkalemia, pericarditis, or fluid overload, the current objective should be to anticipate and prevent, rather than treat, uremic complications. This latter, more aggressive approach finds support in several imperfect but very suggestive studies,[17,18] which, when combined with the concensus impression after two decades of dialysis, suggest that early and frequent dialysis improves healing, permits better nutritional therapy, aids the general clinical state, and facilitates the fighting of infection. Thus, we generally do not permit the creatinine to exceed eight to ten mg/dl or the BUN to exceed 100 mg%. Whether even more agressive dialysis would further improve the outcome remains to be

determined.

Though the role of hyperalimentation is not unequivocally established in ARF, it seems reasonable, on the basis of both prospective[19] and retrospective[20] data to recommend its use. It seems to be particularly of benefit in patients who are markedly catabolic and have such adverse prognostic features as oliguric ARF, pneumonia, gastrointestinal hemorrhage, and generalized sepsis. At this time, the optimal composition of the hyperalimentation fluid, and whether or not it should include non-essential amino acids, has not been rigorously defined.[21]

Conclusion

Despite the advent of hemodialysis, better nutritional therapy, and improved antimicrobial agents, ARF still exacts a high mortality in patients who are otherwise salvageable. Recently available retrospective data [20]has reemphasized the gravity of advanced age and multiple complications in ARF. More importantly, however, this data demonstrated that no single factor or combination of factors predicted a fatal outcome. Thus, we must regard ARF in even the most critical patient as a situation with potential for the ultimate reward—patient survival and restoration of renal function.

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Radiology Continued

Answer: Obstruction of the cystic duct by a gall stone, with hydrops of the gallbladder.

This patient's ultrasound shows thickening of the gallbladder wall, and a transverse diameter of the gallbladder which is at the upper limits of normal. In addition, there is a very dense collection of echos at the gallbladder neck (large arrow) which produce acoustic shadowing (drop-out of echos behind a structure), the hallmark of cholelithiasis (small arrows). The combination of findings suggests that a stone has impacted in the cystic duct, producing dilatation of the gallbladder (hydrops).

The ultrasonic diagnosis of gall stones can be divided into several categories:

1. When shadowing opacities within the gallbladder which move to the dependent protion of the gallbladder are seen, the diagnosis of gall stones is virtually 100% accurate.

2. When the lumen of the gallbladder cannot be identified but high level echos in the region of the gallbladder fossa can be seen, the diagnosis is almost as certain.

3. Non-shadowing opacities within the lumen of the gallbladder which move into the dependent portions at various rates are a finding which is less reliable. This appearance, called "sludge," can be due either to thick inspissated bile or cholesterol crystals in patients who have been fasting or on I.V. fluids for periods of time, multiple tiny calculi smaller than 1 mm. in diameter, or to inflammatory material secondary to an abscess.

4. Increased thickness of the gallbladder wall (greater than 2mm.) has been discussed as a sign which correlates

State of the Art

Antiinflammatory Drugs and Gastric Erosive Disease

In Patients with Arthritis

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Antiinflammatory drugs play a central role in the management of most forms of arthritis. Gastritis, gastric ulcer and bleeding are common complications of this treatment. These side effects are a major problem in arthritis patients, especially those with rheumatoid arthritis who are already at increased risk for developing peptic ulcer disease.[1] It is well known that aspirin can cause gastric ulceration and bleeding.[2-5] Regular use of aspirin in arthritis patients has often led to a variety of endoscopically documented gastric erosive lesions which are often difficult to treat.[6,7] In an attempt to diminish the gastrointestinal side effects accompanying aspirin treatment, various non-steroidal antiinflammatory drugs (NSAID) have recently been developed. Although these drugs were initially thought to not damage the stomach, recent reports show that indomethacin, phenylbutazone, ibuprofen, naproxen, fenoprofen, etc., cause gastric mucosal injury, albeit to a lesser degree than salicylate.[8-11]

We have recently encountered several patients with chronic arthritis who developed significant gastric problems while being treated with either aspirin or NSAID. In this paper, we will briefly report five such cases, discuss current concepts regarding the pathogenesis of gastric mucosal damage by aspirin and NSAID and present an approach to management.

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pathologically with chronic inflammation. To this date, no large series has yet carefully correlated wall thickness in the 3-5 mm. range with pathologic findings. Wall thicknesses greater than 5 mm. seem to correlate well with inflammatory changes in and about the gallbladder wall, and with overt hydrops.

Technical advances within the last several years have made gray scale ultrasound a frequently used modality of first diagnosis in gallbladder disease. With the use of high resolution real-time equipment by experienced personnel, practically all gall stones can be detected. Although the test is highly specific when stones are visualized, it provides only anatomic rather than physiologic information, such as is available via the oral cholecystogram or the rapidly evolving studies using derivatives of iminodiacetic acid.

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Case 1

E.C. is a 67year old white female with osteoarthritis hospitalized because of hematemesis and orthostatic hypotension. She was well until one week prior to hospitalization when she developed right hip and lateral thigh pain. She treated herself with low dose aspirin without relief and was later seen by her physician who recommended increasing the dosage. Soon after beginning 20 aspirin/day, she noted headaches, nausea, tinnitis and severe epigastric distress. One day prior to admission she vomited fresh blood with clots and became dizzy when she stood. On physical examination, an area of hyperesthesia was noted over the right lateral thigh. A herpes zoster eruption developed five days later. Hemoglobin was 12.5 gm %, hematocrit 33.0 vol % with normal red cell indices. Endoscopy showed a 1 X 3 cm ulcer on the posterior aspect of the greater curvature of the stomach. Aspirin was discontinued and she was treated wtih antacids and oral cimetidine. Repeat endoscopy six weeks later demonstrated ulcer healing.

Case 2

P.F. is a 70 year old white female with osteoarthritis who was treated with aspirin, 10 grains qid for several months. When a stool hematest was positive for occult blood, she switched to naproxen. Upper GI radiographs showed tertiary esophageal contractions without other abnormalities. A barium enema was normal. Six months later the stool remained positive for blood. Upper GI endoscopy demonstrated severe. antral gastritis and duodenitis with superficial gastric ulcerations. The naproxen was discontinued and she was treated with cimetidine and antacids. Her stools became negative for occult blood two weeks later.

Case 3

J.B. is a 66 year old Navajo male who was hospitalized because of active upper GI bleeding. The patient was an alcoholic who had a past history of rheumatic heart disease and was hospitalized one month previously for mitral and aortic valve replacement. Postoperatively, he developed intermittent atrial fibrillation and was treated with coumadin. Just prior to discharge, he developed an acute gouty attack in his left knee, which was treated with indomethacin, 50 mg qid. He was discharged with instructions to discontinue indomethacin after two days. However, he continued to take this drug until the current admission.

At the time of hospitalization, his BP was 70/0 and hematocrit 28 vol %. Endoscopy showed a 1.5 cm gastric ulcer on the lesser curvature surrounded by intense gastritis. He was treated with intravenous cimetidine and vitamin K but continued to bleed requiring 10 units of packed red blood cells and five units of fresh frozen plasma over the next two days. A wedge resection of the ulcer was performed. His postoperative course was uncomplicated and he was discharged three weeks later. Further attacks of acute gout were treated with colchicine and he was placed on allopurinol.

Case 4

M.K. is a 63 year old white female with rheumatoid arthritis who was hospitalized for worsening of her joint symptoms and evaluation of a hypochromic microcytic anemia. She had no gastrointestinal symptoms. Her medications included ibuprofen 800 mg qid, chlorambucil, 2 mg bid and prednisone, 5 mg bid. Laboratory data included hematocrit 27 vol %, hemoglobin 8.5 gm %, MCH 22, MCV 71, and stool positive for occult blood. Upper GI radiographs and barium enema were normal. Endoscopy demonstrated mild gastritis and a 0.5 cm pre-pyloric punched out ucleration. Ibuprofen was discontinued and prednisone maintained. She was treated with cimetidine and antacids. Her stools become negative for occult blood over the next few days and she was discharged. Six weeks later a repeat endoscopy showed resolution of the gastritis and complete healing of the ulcer. Ibuprofen therapy was reinstituted and cimetidine continued at 300 mg bid. Bleeding has not recurred.

Case 5

P.R., a 46 year old black woman with a 12 year history of severe rheumatoid arthritis presented with epigastric pain worsening over a two day period. Three weeks previously, she had experienced similar but milder epigastric discomfort and consulted her physician who found her to be anemic with occult blood in the stool. Upper GI radiographs performed the following morning were normal. Her arthritis had been under fair control with prednisone 7.5 mg daily and aspirin 10 grains qid. Her physician discontinued these drugs, and the patient was then started on ibuprofen 400 mg qid. However, abdominal pain persisted and she was hospitalized when her hematocrit was found to be 28 vol %. Physical examination showed blood pressure 140/90, and pulse 92/minute without orthostatic change. There was epigastric tenderness and blood in the stool. Upper GI endoscopy demonstrated a small hiatus hernia, marked antral gastritis with punctate superficial ulcerations and a 1.5 cm ulceration on the lesser curvature of the stomach. Ibuprofen and prednisone were discontinued and the patient was started on cimetidine, 300 mg qid. After three days, the abdominal pain persisted and her joint symptoms had increased markedly. Because of worsening arthritic symptoms and a concern that the abdominal pain might be due to relative adrenal insufficiency, prednisone was restarted. She subsequently improved. Follow-up endoscopy in eight weeks demonstrated complete healing of the ulcer.

Discussion

A variety of drugs have been suspected of causing erosive injury to the stomach. Drugs which have been most frequently implicated include reserpine, caffeine, corticosteroids, salicylate, and recently NSAID such as indomethacin, phenylbutazone, naproxen, ibuprofen, fenoprofen, etc.[12] The frequent occurrence of peptic ulcer symptoms, superficial erosive gastritis and gastric

ulcer in patients treated with antiinflammatory drugs had led to the clinical impression of a causal relationship.

Drug-related gastric damage and epidemiological data concerning this problem have been extensively and critically reviewed by Cooke.[11-13] Caffeine and alcohol stimulate gastric acid secretion but do not cause peptic ulcer. Also, no definite link can be made with reserpine. Current evidence does not support the widely held belief that adrenocorticosteroids cause ulcer disease.[14,15] Aspirin, however, has been shown conclusively to cause gastric ulcer and gastritis with bleeding; aspirin does not, however, cause duodenal ucler.[4,10,11,16] Ethanol ingestion enhances the effects of aspirin on gastric mucosa and increases blood loss.[17] The incidence of ulcer disease with NSAID is somewhat less clear. However, there are clinical reports of gastric mucosal damage.[12] Endoscopic studies in normal volunteers treated with NSAID have shown gastritis and erosions.[9,10] Whether NSAID cause gastric ucler with less frequency than aspirin is not known, although clinical experience shows that these drugs are often better tolerated than aspirin.

Most theories of drug-induced gastric ulceration invoke a drug-induced change in gastric acid secretion and/or a change in gastric mucosal resistance. None of the drugs studied to date, with the exception of alcohol and caffeine have significant effects on gastric acid secretion. However, it has been shown that gastric acid can damage mucosa of the stomach without being present in excessive amounts. The mechanism for this effect was first described by Davenport who demonstrated a functional mucosal barrier which prevents hydrogen ions in the gastric lumen from back-diffusing into the mucosa.[18] This barrier to acid back-diffusion is manifest in part by the electrical potential difference maintained across the mucosa. The barrier can be broken by ischemia and by a variety of drugs including aspirin and ethanol.[19] When the barrier is broken, gastric acid diffuses into the mucosa causing additional damage and a fall in the gastric mucosal potential difference. Of all drugs which have been evaluated *in vitro* for their effect on the gastric mucosal barrier to acid back-diffusion, only unbuffered aspirin and ethanol consistently break this barrier. In animal studies indomethacin has an inconstant effect.[20] Naproxen, ibuprofen, and fenoprofen also do not have a constant effect on the mucosal barrier. They are not considered to cause mucosal damage by this mechanism.[12] In clinical studies, only aspirin and ethanol but not indomethacin or phenylbutazone have been shown to cause a decrease in potential difference across the gastric mucosa, reflecting a reduction in the mucosal barrier.[21,22]

Another postulated mechanism for gastric mucosal injury has been a drug-related effect on mucosal prostaglandin synthesis and content.[23] Most of the NSAID inhibit prostaglandin synthesis. Prostaglandin E^2 and its methyl analogs inhibit gastric acid secretion possibly by increasing intracellular cyclic AMP.[24] In animal studies, oral administration of prostaglandin E_2 along with indomethacin or aspirin prevented the gastric erosion and ulceration which developed with use of these drugs alone.[25]

Apart from a direct effect on the mucosa, antiinflammatory drugs may also alter the quantity and quality of gastric mucus. In dogs, aspirin, phenylbutazone and indomethacin decrease the rate of secretion of gastric mucus and change its carbohydrate composition such that the activity of proteolytic enzymes is increased.[26] Gastric blood flow also appears to play a role in both gastric acid secretion and mucosal resistance. Reduced gastric blood flow is associated with diminished mucosal resistance. In dogs, both aspirin and indomethacin decrease gastric mucosal blood flow. This effect may also be mediated through prostaglandins.[27]

Most of the work aimed at investigating the mechanism of deleterious drug effects on gastric mucosa has been done using salicylate. The results of a considerable number of studies have recently been reviewed by Fromm.[28] It is clear that acid must be present in the stomach for mucosal injury to occur. Salicylates initially appear to selectively increase permeability of the gastric mucosa to cations permitting diffusion of luminal hydrogen ion into tissue. This, in turn, induces a major and more non-specific increase in membrane permeability leading to significant tissue damage. Salicylates can also interfere with various metabolic processes in gastric mucosa.[29,30]

Recently, endoscopy and histology of gastric mucosa have become the major techniques employed to evaluate drug-induced damage to the stomach. Upper GI radiography is too insensitive. Endoscopic studies have documented extensive gastric abnormalities in patients taking both aspirin and various NSAID.[5,10-12] The most severe damage is usually produced by unbuffered aspirin. Buffering the aspirin reduces mucosal damage, but the protective effect is not complete. In human volunteers taking large doses of aspirin, visible mucosal changes occur primarily in areas adjacent to small particles of undissolved aspirin. Early histological changes which are present at 10 minutes include abnormal staining, cytoplasmic vacuoles and some loss of the apical portion of gastric mucosal cells. Later changes at 30 minutes include further destruction of the apical cell area with cell sloughing, giving the mucosal surface an honeycomb appearance by scanning electron microscopy.[31] Increased cell sloughing is reflected by an increase in the DNA content of gastric washings. Electron microscopic studies show no effect on tight junctions between gastric epithelial cells. There appears to be complete recovery of all histologic abnormalities one to six hours after salicylate administration.[31] Unfortunately, such extensive histologic investigations have not been done in man with the NSAID. In a clinical study using a group of normal volunteers, Lanza, et al, found endoscopic evidence for gastric mucosal damage in varying degrees of severity with all NSAID tested.[9,10]

Importantly, significant mucosal injury was often present in asymptomatic subjects. The most severe gastric mucosal damage was caused by high dose aspirin followed by high dose (750 mg/day) naproxen then by both doses (100 and 150 mg/day) of indomethacin, with the least damage being caused by low dose naproxen (500 mg/day) and ibuprofen (1600 mg/day). This study suggests that there may be a gradient in incidence and severity of gastric mucosal injury with different NSAID and that severe gastric mucosal damage may be present in the absence of symptoms.

It has been suggested that gastric mucosal damage associated with antiinflammatory drug treatment may be lessened or prevented by the use antacids and cimetidine.[13,31-33] Indeed, in patients with rheumatoid arthritis, it has recently been demonstrated that small (less than 1.0 cm) "aspirin-induced" gastric ulcers heal despite continued aspirin treatment if antacid therapy is given.[34] However, this study involved only a small number of subjects in a highly controlled situation and further studies are needed before conclusions regarding clincial application are warranted. Nevertheless, this study made an important point about the mechanism of aspirin-induced gastric injury and certainly gives direction to further efforts at treatment. Although NSAID do not specifically break the barrier to acid back-diffusion, mucosal damage with these agents may be a secondary phenomenon, thus giving some rationale for the use of antacids and H_2-receptor blocking drugs like cimetidine. An intriguing new theapeutic possibility has come from observations relating to the protective effect of prostaglandins on gastric mucosa.[24,25] A recent study demonstrated in rats that prostaglandin E_2 had a greater protective effect against thermal, acid and alkali-induced gastric mucosal injury than did pre-treatment with cimetidine.[36] Since gastric mucosal damage with NSAID appears to be related to a reduction in mucosal prostglandins, it is tempting to speculate that if an oral preparation of PGE_2 becomes available for clinical use, it could have application in the treatment of gastric mucosal injury from NSAID.

Based on the mechanisms of drug-induced gastric mucosal damage and clinical experience, we recommend discontinuing both aspirin and NSAID in patients where definite mucosal ulceration is present. Intensive treatment with antacids and/or cimetidine should be started and continued for at least six to eight weeks. Ulcer healing should be documented endoscopically prior to restarting antiinflammatory drug therapy. In this situation, we recommend continuing low dose antacid therapy after meals and at bedtime and/or cimetidine twice daily. In patients with only mild gastritis, reduction of aspirin or NSAID dosage plus antacid treatment is often sufficient. Prophylactic treatment with cimetidine and/or antacids may have a role in management of the high risk arthritis patient who has previously documented peptic ulcer or is being placed on high doses of aspirin or NSAID. However, the value of this remains to be documented by controlled studies.

In the patient with active arthritis, discontinuation of NSAID may result in a flare of disease. Analgesics and physical therapy, e.g., hydrocolator pack applications and Hubbard tank immersion, may help alleviate pain. If the patient is taking a remission-inducing agent such as gold or penicillamine, it should be continued. If the patient is not on such a drug, one should be added to the therapeutic regimen in an effort to eventually control disease activity. Since there is no evidence that corticosteroids are ulcerogenic, steroid drugs should be maintained and perhaps increased to control disease activity and prevent adrenal insufficiency under stress. In individual cases, such as a patient with a severe flare in arthritis symptoms, it may be necessary to use steroids for the first time during the period when NSAID are withheld.

Conclusion

We have presented five examples of arthritis patients who developed symptomatic gastric mucosal disease secondary to treatment with antiinflammatory drugs. Clinical presentations varied from major GI bleeding to asymptomatic anemia. Case 1 illustrated a major GI bleed during high dose aspirin, while Case 2 was an example of occult GI bleeding occurring with NSAID treatment. Additive effects of alcohol and coumadin with indomethacin almost led to disasterous results in Case 3. An unexplained microcytic hypochromic anemia was the only clue to drug induced gastric pathology in Case 4. Finally, Case 5 underscores the importance of corticosteroids in the management of gastric disease in steroid-dependent arthritis patients.

The mechanisms for gastric injury caused by such drugs have been reviewed and the important role of gastric acid in producing clinically significant mucosal injury has been emphasized. Based on these concepts, appropriate treatment of this common clinical problem involves stopping the offending drug and storing pharmacological attempts to reduce the effect of gastric acid. Corticosteroids should not be discontinued and occasionally should be increased. Once the gastric mucosal lesion has healed, aspirin or a NSAID can be reinstituted. However, in this case, antacid therapy should be continued.

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Correspondence

Editor—

I read with interest the article in February, 1980 issue of *Arizona Medicine* entitled "Foreign Body Aspiration in Childhood," by Drs. Logvinoff and Taussig. I agree with all their main points, and it is for that reason I feel so strongly about the unfortunate nature of their opening statement.

Their opening statement reads, "Foreign body aspiration is the most common cause of accidental death in healthy children." This is an obvious falsehood. The Arizona Department of Health Services has recorded 155 accidental deaths in children under age 12 in 1978. Sixty-one deaths in this age group were due to auto accidents, 40 were from drowning, 17 were from burns and seven were from foreign body aspiration. My own experience agrees with these Arizona State figures. Death in children from foreign body aspiration is rare. I believe that this inacurate opening statement casts an unfortunate shadow over the quite worthy remainder of the article.

Sincerely yours,

Joseph T. Zerella, M.D.
Phoenix, Arizona

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Defining Excessive Fees

There are some items that I wish to bring to the attention of the general membership. The first has to do with my testimony before the Board of Medical Examiners June 5. This was a public hearing. I was there representing the Arizona Medical Association.

First, some background material. At the ArMA Executive Committee meeting Friday evening, May 30, we learned this public hearing on a proposed rule defining excessive fees was to take place. The proposed rule change comes under the section defining fraud and states: *"Fraud is gross, willful or continued overcharging for professional services or charging for services not rendered or misrepresentation, deceit or trickery in any act, document or record relating to medical practice."*

Now BOMEX already has a rule dealing with fees charged by physicians. This rule is, in effect, Section 7 of the AMA's Principles of Medical Ethics: *"A physician's fee should be commensurate with the service rendered and the patient's ability to pay."* However, the BOMEX Board wanted a more explicit rule defining excessive fees to assist them in dealing appropriately with the occasional physician brought to their attention for charging grossly excessive fees.

At the beginning of the public hearing Dr. George Scharf, BOMEX Chairman, stated clearly that it was **not** the Board's intent to set fees. He explained Board members do need better guidelines to deal with the physician who grossly overcharges.

What is a "gross overcharge"? Is $495.00 to repair a small laceration that does not go through the fascia too much? Is $2,000.00 for a hemorrhoidectomy excessive when five other surgeons reviewed the operative notes and came up with a suggested fee of $1,000.00?

These cases are few and far between but they are the kinds of questions sometimes confronting BOMEX. It's a complex problem and the Board is willing to accept our help in trying to find the answer.

Some of my colleagues say fee review is not within the purview of the Board of Medical Examiners. Some say that disciplining of physicians should be left solely in the hands of the County Medical Societies and ArMA.

I would agree in those cases where the wayward behavior of the doctor can be corrected by peer pressure. But what if the doctor does not belong to organized medicine? At the present time, we have 1,700 licensed and practicing Arizona physicians who fall into this category. What about those members who are not influenced by peer pressure and continue to charge ridiculous fees? What about the physician who sees medicine as a business and charges what the traffic will bear?

The essence of my testimony before BOMEX was that the proposed rule as presently drafted would not accomplish its intended objectives. That it was dangerously vague and ambiguous. That it did not establish workable guidlines for the Board. Furthermore, that the term "fraud" should be stricken from the rule as being unnecessarily inflammatory.

Our position was that the focus of the rule should be on a consideration of a variety of factors that lead to the setting of an appropirate fee. We offered the services of ArMA in assisting BOMEX in formulating a more workable rule.

BOMEX voted to refer the proposed rule to a subcommittee of its members for further study. This matter may be discussed at a subsequent public hearing and I need your input.

Medical Malpractice Panels

The March 31 issue of the *AMA Newsletter* (green sheet) tells of the legal attacks on these panels in several states.

I have recently learned that the panels in Arizona are having the same problems. Just the other day, I had a long conversation with Pima County Superior Court Judge J. Richard Hannah. He told me of the problems that he has in convening a panel. First, setting a time and date for the panel to meet—it is difficult to get attorneys and doctors together. Then, finding doctors of the same or similar specialty to come forth and testify.

We must remember that these panels were adopted by the Arizona State Legislature at the behest of the medical profession and for the protection of physicians. It is incombent upon us as physicians to make this process work smoothly.

If you are called to give testimony, I ask you to acquaint yourself with all aspects of the procedure and be thoroughly familiar with the role you are to play. Furthermore, make every attempt to meet with the panel as soon as possible. If we doctors fail to make this system effective, these review panels may be discarded.

Long Range Planning for ArMA

I have appointed John Oakley of Prescott as Chairman of the Committee on Long Range Planning. He will be

selecting his committee members soon so that the committee can convene and get under way.

In discussing the goals of the Long Range Planning Committee at the last Executive meeting, the question of unified membership came up once again. John indicated that the members in the rural counties were particularly irate at the results of the last vote on this issue.

Some delegates from Maricopa, and well as Pima, were absent from the House of Delegates for the vote. Since this was a bylaws change, we needed a two-thirds affirmative vote of the delegates "present and voting." The bylaws change failed by approximately 13 votes.

I abstained from voting on the issue. I felt there were strong points both pro and con; therefore I, as your president, should maintain a neutral stance.

I wish this issue could be resolved so that it would no longer be so devisive. I can assure you that the Executive Committee is looking into ways to do this and I will keep you informed.

1981 Convention

The Board of Directors is investigating the possibility of moving the dates of the 1981 annual meeting to the last week in May in order to get more favorable hotel room rates.

October Meeting of Board of Directors

This meeting will be held at 10 a.m., Saturday, October 4, in Tucson. The Executive Committee will be meeting in Tucson the previous evening. After the Board concludes its meeting Saturday, we will have a luncheon with members of the Medical School staff at the Arizona Inn. The purpose of this luncheon is to enable ArMA Board members and Medical School faculty to meet informally and get better acquainted. Then, at 2:30 p.m. there is planned an "official family meeting" of ArMA officers, Board members and committee chairmen with an officer of the AMA. The national representative will give us an update on current AMA activities.

Remember that the other ArMA officers and I are always glad to receive feedback from any of the members. Should any member, or group of members, wish to discuss any issue with the Executive Committee they are welcome to do so. Call me for the time and place of the next meeting.

You will be hearing from me again next month.

John T. Clymer, M.D.
President, ArMA

The Mucio F. Delgado Clinic for Neuromuscular Disorders

The Clinic for Neuromuscular Disorders is a function of the Department of Neurology at the Arizona Health Sciences Center established with the support of the Muscular Dystrophy Association in 1972. Currently under the direction of Dr. Lawrence Z. Stern, it serves Tucson and neighboring communities as well as being a referral center for the entire state of Arizona. The case load increase rate during its first six years exceeded that of any other MDA clinic in the country, which now number well over 200. In October 1978, in a joint action of the Board of Directors and the Arizona Board of Regents, the clinic was renamed and dedicated by Jerry Lewis, MDA's National Chairman. It was renamed to honor the memory of "Moose" Delgado, a distinguished University of Arizona alumnus who served as National Director of Program Development of MDA.

The MDA clinic provides services to individuals with 40 different neuromuscular diseases who live in southern Arizona and, upon referral, from anywhere in the United States and its territories. These diseases include not only the various forms of muscular dystrophy but also the spinal muscular atrophies, amyotrophic lateral sclerosis (ALS), inflammatory myopathies (such as polymyositis), diseases of peripheral nerve, diseases of the neuromuscular junction (such as myasthenia gravis), the myotonias, metabolic diseases of muscle, myopathies due to endocrine abnormalities and less common muscle diseases including malignant hyperthermia, a hereditary disorder that may produce a severe reaction to certain anesthetic and muscle relaxants used with anesthesia.

Clinic services are provided free of charge to patients suffering from any of the above disorders, as well as for the initial diagnostic evaluation of any patient suspected of having one of MDA's covered illnesses. This initial diagnostic examination includes all necessary tests including electromyography (EMG) and nerve conduction studies, biopsy and hospitalization up to a maximum of three days when indicated. Other clinic services include carrier detection (when possible), medical consultations, electrocardiography (EKG), pulmonary and muscle function tests, genetic counseling and social services. Other patient and community services include orthopedic aids, respiratory equipment, also physical, occupational and respiratory therapy. Flu inoculations and transportation of patients for medical appointments, summer camps and other chapter sponsored recreation are also provided.

The Mucio F. Delgado Clinic also serves as the focus for a broad-based multidisciplinary clinical research program and an MDA-sponsored fellowship training program in the field of neuromuscular disease. Clinic is held two half days per week. Patient appointments at the clinic may be made through the MDA office in Tucson.

Lawrence Z. Stern, M.D.
Associate Professor of Neurology
Louis J. Kettel, M.D.
Dean
College of Medicine

Erratta

The authors of the article *A Program of Primary Care Education at the University of Arizona*, which appeared in the March issue Vol. 37, No.3 page 185, were Antia Stafford, M.D., Associate Professor and Director, Ambulatory Section; Helen Johnson, M.D., Staff Pediatrician, Department of Pediatrics and Louis J. Kettel, M.D., Dean, College of Medicine.

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WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.. A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

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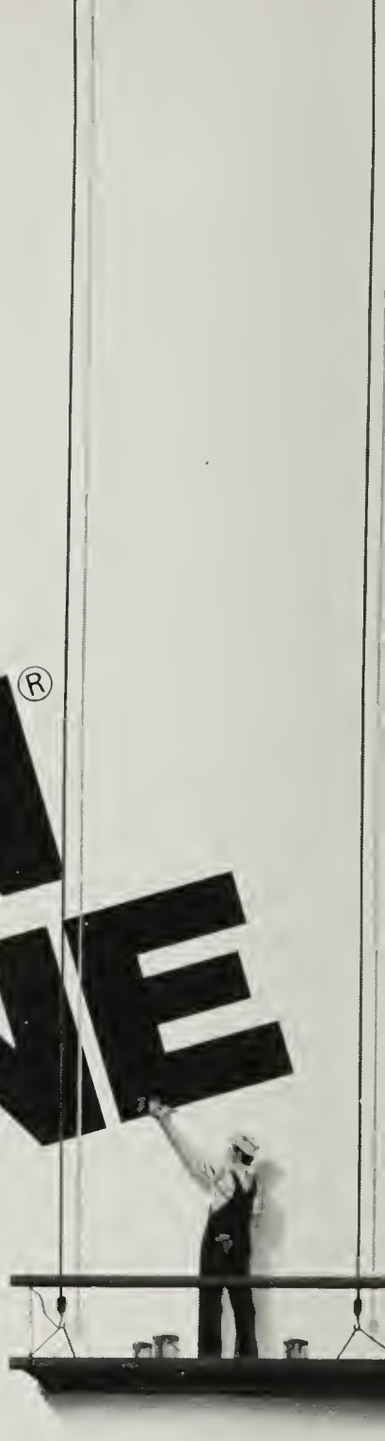
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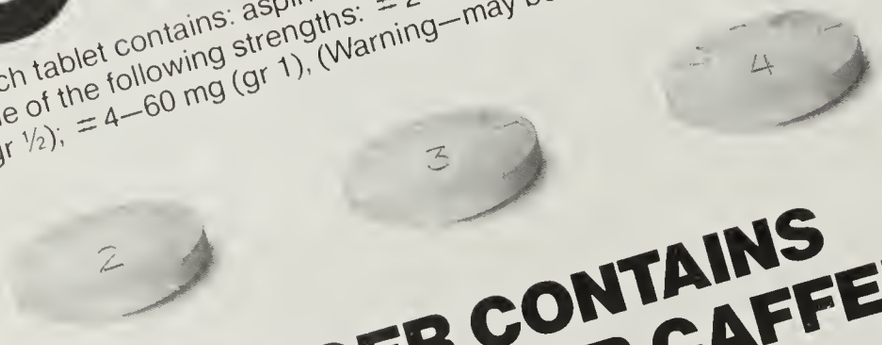
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Auxiliary Highlights

New President Installed

Mrs. Lynn David Newman (Joyce) of Glendale was installed as President of the Arizona Medical Association Auxiliary at its 50th anniversary meeting in Phoenix in April. Mrs. George T. Hoffmann (Janet) of Phoenix was named President-Elect.

Other new state officers are Mrs. Richard Collins (Mary Lee) of Scottsdale, First Vice-President; Mrs. Albert Rosenblatt (Anita) of Kingman, Second Vice-President; Mrs. Harry Tennant (Ann) of Yuma, Secretary; and Mrs. Richard Morgan (Carolyn) of Phoenix, Treasurer. Mrs. Orlin Wry, Jr., (Connie) of Tucson, Mrs. Joe McNally (Sue) of Prescott and Mrs. John Clymer (Eloise) of Tucson serve as Directors.

Joyce Newman, a registered nurse, has worked with the Auxiliary since coming to Arizona from Houston, Texas, with her obstetrician-gynecologist husband. She served the Maricopa County Auxiliary as liaison with the interns' and residents' wives group, GEMS chairman, and treasurer for two years. She was Public Relations Committee chairman and First Vice-President and Membership Committee Chairman for the ArMA Auxiliary before becoming President-Elect in 1979-80.

Joyce has twice been a delegate to the Auxiliary's national convention and has attended its Fall Confluence in 1979, Regional Meetings in 1979 and 1980, and AMA's National Leadership Conference in 1980.

The Newman's have four children so Joyce has been involved in Camp Fire, Boy Scouts, Girl Scouts and Sunday School. She has just completed a term as President of the Horizon School PTA.

In other action at the annual meeting, the Auxiliary voted to increase state dues to $12. Inflation was the major reason for the increase but the additional revenue will permit the Auxiliary to expand its health education programs, leadership training, membership recruitment, legislative and HSA monitoring.

Joyce has increased the size of the state board in an effort to involve more Auxiliary members from all parts of the state.

Her goals for the Auxiliary this year include:

An emphasis on getting to know each other at the Fall Conference, which will be held September 23-24 at Poco Diablo Resort in Sedona.

Continued speakers' training for Auxiliary members and doctors.

Publication of an Auxiliary Handbook, listing program resources and other information helpful to state and county committees.

Leadership training for board members of state and county Auxiliaries.

Stimulation of good health education in the public schools by encouraging the use of "Early Start to Good Health" and "Health Network" kits in the elementary schools.

Reprinting and distribution of the Auxiliary's pamphlet, "Is He Sick? Shall I Send Him to School?"

In consultation with ArMA, consider developing a cost containment information program for the public based on a Washington Auxiliary film, "The Cost of Caring."

Continue to monitor HSAs and to keep members informed of health legislation issues at the state and national levels.

Mrs. Fred Holmes Honored

Mrs. Fred G. Holmes (Lore) of Phoenix was named to Honorary Membership in the ArMA Auxiliary at the 50th anniversary luncheon. Mrs. Holmes was instrumental in the founding of both the Maricopa and Arizona Auxiliaries.

County Auxiliaries presented certificates honoring outstanding service to Mrs. Joseph Reno, Coconino; Mrs. Clare Johnson, Maricopa; Mrs. John Standifer, Mohave; Mrs. James L. Parsons and Mrs John Clymer, Pima; Mrs. Melvin Phillips, Yavapai, and Mrs. Charles Matheus, Yuma.

More Funds for Medical School

ArMA Auxiliary made two substantial gifts to Arizona's College of Medicine at the 1980 annual meeting.

Mrs. William Bishop of Globe, chairman of the Hamer Loan Fund Committee, gave Dean Louis Kettel a check for $6000 to be added to the Auxiliary's revolving emergency loan fund for medical students. The fund now totals $22,000. No interest is charged for the loans, which are administered by the medical school.

Mrs. Ronald Johnson of Phoenix, chairman of the AMA-ERF Committee, presented a check for more than $18,000 to Dr. Kettel at the ArMA President's Banquet. The unrestricted gift represents money donated by Arizona physicians and money raised by the county medical Auxiliaries.

Not content, the AMA-ERF committee raised another $350 for the medical school at the convention itself. That sum will be included in next year's unrestricted gift.

Mrs. John T. Clymer

ARMA

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ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held in South Court Room No. 2 at The Pointe Resort, 7677 North 16th Street, Phoenix, Arizona, on Wednesday, April 23, 1980, a quorum being present, convened at 6:31 a.m., William E. Crisp, M.D., President and Chairman, presiding.

Arizona Department Of Health Services

Maternity Care Program

Dr. Dandoy advised the committee that as a result of recent efforts in legislature the Department had been informally advised that it would recieve new program funding for any maternity care activities of the Department during the year.

Rubella Regulation

As an update, Dr. Dandoy reported that the recent regulation regarding rubella immunization had been withdrawn and would be the subject of further evaluation, study and discussion at an upcoming meeting to be held in Georgia.

Emergency Medical Services

The committee was briefed by Dr. Dandoy on emergency medical services within the state and the fact that the Department of Health Services was recommending that it, as opposed to the Department of Public Safety, be vested with all emergency medical services planning, coordination, regulatory, management and funding responsibilities. Dr. Dandoy reported that present Arizona law provided that these functions be handled by DPS and, therefore, would necessarily require legislative decision.

Border States Health Research Synthesis and Dissemination Project

Letter from Andrew W. Nichols, M.D., dated March 24, 1980 *Received.*

Review of Board of Directors' Agenda for April 23, 1980

The agenda for the Board of Directors' meeting on April 23, 1980 was discussed, item by item, with additions and changes being made.

Review of House of Delegates' Agenda

The committee reviewed and discussed the agenda for House of Delegates' meetings scheduled for April 23 and April 25, 1980.

Other Business

1980-81 Meeting Schedule

It was moved and carried approving the following list of tentative dates for meetings of the Executive Committee during 1980-81:

May 30, 1980	Friday	6:30 p.m.
June 27, 1980	Friday	6:30 p.m.
August 8, 1980	Friday	6:30 p.m.
September 19, 1980	Friday	6:30 p.m.
October 3, 1980	Friday	6:30 p.m.
November 21, 1980	Friday	6:30 p.m.
December 19, 1980	Friday	6:30 p.m.
January 9, 1981	Friday	6:30 p.m.
February 27, 1981	Friday	6:30 p.m.
March 20, 1981	Friday	6:30 p.m.
April 22, 1981	Wednesday	6:00 a.m. - Annl Mtg.

Bomex Space

The committee was advised of the potential lease of the recently vacated BOMEX space in the ArMA building for use in training of emergency medical technicians and that, hopefully, this matter could be finalized the first part of May. Mr. Robinson also advised the members that interest had been indicated by the INA Health Plan and would be considered if negotiations on the first offer were unable to be concluded.

Gary Barnett reported on the final days of the legislature, specifically advising the committee of the passage of the homeopathy bill, providing for a Board of Homeopathy within the state, and the passage of the optometric "eye drop" bill, on which there was an indication that some type of legal action might be filed. Mr. Barnett further advised that, on both of these bills, ArMA had made written request of the Governor for veto.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held in North Court Rooms #1 and #2, The Pointe Resort, 7677 N. 16th Street, Phoenix, Arizona, on Wednesday, April 23, 1980, a quorum being present convened at 8:02 a.m., William E. Crisp, M.D., President and Chairman, presiding.

Board of Directors

Conciliation Agreement

Finalized ArMPAC/FEC Conciliation Agreement *Received.*

Executive Committee

Ratification of Minutes

It was moved and carried to approve and ratify the minutes of the meeting of the Executive Committee held on March 7, 1980.

Membership Classification Changes Approved

Coconino County Medical Society

1. Jay L. Sitterley, M.D. - Active to Associate -Account Retirement - Dues Exempt - Effective 3/1/80

Gila County Medical Society

1. Alexander J. Bosse, M.D. - Active to Associate -Account Retirement - Dues Exempt - Effective 1/1/80 -including refund of ArMA 1980 dues and request of AMA for refund of 1980 dues.

Maricopa County Medical Society

1. John S. Baker, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80 - including refund of ArMA 1980 dues and request of AMA for refund of 1980 dues.
2. Raymond J. Donovan, M.D. - Active to Associate -Account temporarily out of practice - Dues Exempt -Effective 1/1/80.
3. Joseph S. Lentz, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/80.
4. Joe C. Ehrlich, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.

Pima County Medical Society

1. Geraldine L. Freeman, M.D. - Active to Associate -Account Retirement - Dues Exempt - Effective 1/1/80.
2. Ray O. Hatch - New Medical Student - Effective 4/23/80.
3. William L. Martin, M.D. - Active to Service - Account Air Force Physician - One Quarter Dues - Effective 4/23/80.
4. David Rifkind, M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 1/1/80.

Pima County Medical Society

1. James T. O'Neil, M.D. - Active to Associate -Account Retirement - Dues Exempt - Effective 1/1/80.

Yuma County Medical Society

1. George O'Brien, M.D. - Active to

Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.
2. Dudley A. Reekie, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.

Additional Student Memberships Approved

1. Toni Brayer
2. Bob England
3. Enrique Fernandez
4. Rick Paicius
5. John VanDeventer

Finance Committee

Financial Statement

Financial Statement for the period ending 3/31/80 *Received.*

Grievance Committee

Principles of Medical Ethics

The board reviewed the recommendation of the Grievance Committee relating to amendment of the AMA Principles of Medical Ethics and also considered a revision of those Principles prepared and submitted by that committee following a lengthy discussion,

It was moved and carried that the Board of Directors present to the House of Delegates During its meeting scheduled on April 23, 1980, the following resolution regarding the principles of medical ethics:

"**Whereas,** After careful review of the various proposals for revision of the Principles of Medical Ethics, the Board of Directors of the Arizona Medical Association feels that the present Principles continue to be adequate, with some minor revisions, therefore be it

Resolved, that the Association support the continuation of the Principles of Medical Ethics as revised in 1957 and presently in existence, with the following modifications: 1) The deletion of the second portion of Section 3 thereof; 2) the deletion of the last sentence of Section 5 thereof; 3) the addition of a Section 11, setting forth in essence, the following language: 'A physician shall not solicit patients, nor shall a physician advertise in a false, fraudulent, deceptive or misleading manner or advertise the quality of medical service,' the edited version to read as follows:

Preamble. - These principles are intended to aid physicians individually and collectively in maintaining a high level of ethical conduct. They are not laws but standards by which a physician may determine the propriety of his conduct in his relationship with patients, with colleagues, with members of allied professions, and with the public.

Section 1. - The principle objective of the medical profession is to render service to humanity with full respect for the dignity of man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion.

Section 2. - Physicians should strive continually to improve medical knowledge and skill, and should make available to their patients and colleagues the benefits of their professional attainments.

Section 3 - Physicians should practice a method of healing founded on a scientific basis.

Section 4 - The medical profession should safeguard the public and itself against physicians deficient in moral character or professional competence. Physicians should observe all laws, uphold the dignity and honor of the profession and accept its self-imposed disciplines. They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession.

Section 5. - A physicians may choose whom he will serve. In an emergency, however, he should render service to the best of their ability. Having undertaken the care of patients, he may not neglect him; and unless he has been discharged he may discontinue his service only after giving adquate notice.

Section 6. - A physicians should not dispose of his services under terms or conditions which tend to interfere with or impair the free and complete exercise of his medical judgement and skill or tend to cause a deterioration of the quality of medical care.

Section 7. - In the practice of medicine, a physician should limit the source of his professional income to medical services actually rendered by him, or under his supervision, to his patients. His fee should be commensurate with the services rendered and the patient's ability to pay. He should neither pay nor receive a commission for referral of patients. Drugs, remedies or appliances may be dispensed or supplied by the physician provided it is in the best interest of the patient.

Section 8. - A physician should seek consultation upon request; in doubtful or difficult cases; or whenever it appears tht the quality of medical service may be enhanced thereby.

Section 9. - A physician may not reveal the confidence entrusted to him in the course of medical attendance, or the deficiencies he may observe in the character of patients, unless required to do so by law or unless it becomes necessary in order to protect the welfare of the individual or of the community.

Section 10. - The honored ideals of the medical profession imply that the responsibilities of the physician extend not only to individual, but also to society where these responsibilities deserve his interest and participation in activities which have the purpose of improving both the health and the well-being of the individual and the community.

Section 11. - A physician shall not solicit patients, nor shall a physician advertise in a false, fraudulent deceptive or misleading manner or advertise the quality of medical service.'

Arizona's Obligatory Reporting Law

The Board reviewed the action of the Grievance Committee taken during meeting on April 11, 1980 relating to a request of the Physician Rehabilitation Committee on possible amendment of Arizona's statute requiring obligatory reporting of impaired physicians, and

It was moved and carried to accept the recommendation of the Grievance Committee that the Arizona Medical Association take no action relative to the recent request of the Physician Rehabilitation Committee relating to a possible Amendment of the Arizona Obligatory Reporting Law.

Maternal and Child Health Care Committee

Arizona Perinatal Program

Activities report re: Arizona Perinatal Program *Received.*

Medical Economics Committee

Mental Health Coverage

Status report re: Position on mental health coverage *Received.*

Medical Education Committee

ArMA/BOMEX CME Requirment

The Board reviewed the action of the Medical Education Committee taken in meeting on April 10, 1980 and, following discussion,

It was moved and carried to accept the recommendation of the Medical Education Committee that the Arizona Medical Association continue to establish the CME requirments for membership by its own standards rather than attempting to tie them into the CME requirements for licensure.

Accreditations

Continuation of Arizona State Hospital CME accreditaiton and granting of provisional CME accreditation to the Arizona Thoracic Society/Arizona Lung Association *Received.*

Other Business

1980-81 Meeting Schedule

It was moved and carried approving the following list of tentative dates for the 1980-81 Board of Director's meetings:

See top of next page

Legislative Update

Gary L. Barnett advised the Board that while the legislature was still in session it had, in what was intended to be its last days, passed several bills in which ArMA had interest. Among these were the

August 9, 1980	Saturday	10:00 a.m.
October 4, 1980	Saturday	10:00 a.m.
January 10, 1981	Saturday	10:00 a.m.
March 21, 1981	Saturday	10:00 a.m.
April 22, 1981	Wednesday	8:00 a.m. Annl. Meeting
April 24, 1981	Friday	12:00 Noon Annl. Meeting

optometric "eye drop" bill, on which a veto by the Governor had already been requested by the Association, and the homeopathic bill providing for a Board of Homeopathy within the state, on which legal counsel had prepared for delivery on April 23, 1980, a like request for veto.

Edward Jacobson, Esq., counsel for the Association, discussed with the Board the indication that there might be some type of litigation filed regarding the optometric bill and reviewed for the members the contents of the letter which was to be delivered to the Governor regarding the homeopathic bill.

Legal Counsel

The Board noted that, as of May 1, 1980, Edward Jacobson, Esq. would have personally acted as counsel for the Association for 30 years. The members extended to Mr. Jacobson their thanks and appreciation for his expert guidance and counsel, as well as time and effort expended by him, to the Association and its staff during that time.

PHYSICIAN REHABILITATION COMMITTEE

The meeting of the Physician Rehabilitation Committee of the Arizona Medical Association, Inc. held in Quad A, Pointe of Order, in the Pointe of View Restaurant, at The Pointe Resort, 7677 North 16th Street, Phoenix, Arizona, on Wednesday, April 23, 1980, a quorum being present, convened at 6:13 p.m., Thomas E. Bittker, M.D., Chairman, presiding.

The Zebra Club

Harrison G. Butler, M.D., and Paul B. Borgesen, M.D. gave the members a report on the development of a Zebra Club in the Maricopa area. They stated that they helped form a group of physicians who are recovering from the disease of alcoholism. The name chosen for this group is the "Zebra Club". The club was started because it was perceived by the physicians of the rehabilitation committee of ArMA that we need a group of recovering alcoholic physicians to act as a referral source for physicians suffering from this disease. The group will be able to take call on a rotating schedule twenty-four hours a day. They are planning to use the phone number of the National Council on Alcoholism who will refer the call to the appropriate recovering physician on call. They are currently limiting referrals to the health care profession who take care of people who are suffering from alcohol addiction or other substance abuse. Dr. Young from Tucson also reported on an anonymity group in Tucson and its current development.

The above discussion led to consideration of the desirability of changing the name of the Physician Rehabilitation Committee. It was determined to find out what other names are being used.

It was moved and carried to continue the study on a name change of the Physician Rehabilitation Committee and, as a part of that study, to obtain a list of all such committees throughout the United States, such list to be circulated to committee members for future discussion.

Committee Size

was generally agreed by the committee that the committee should be increased in numbers so that there would be more contact and coordination physicians available. Several recommendations were made and Dr. Clymer was to be asked to appoint them to the committee.

Second Regional Meeting of Representatives of State Medical Association

Physician Rehabilitation Committee

Dr. Bittker reported on the subject meeting, which was held on February 11 and 12, 1980 in San Diego, California, under the sponsorship of the California Medical Association. Many of the recommendations of that group were reviewed and it was determined that, in regard to supervision and treatment, the Arizona Medical Association Physician Rehabilitation Committee does not offer to supervise the treatment of impaired physicians; that the committee's philosophy is to treat locally when it is possible, but the committee would identify local facilities for treatment is asked.

It was also determined that Ross Landess, M.D. would develop a formal statement of philosophy for treatment.

Arizona's Obligatory Reporting Law

The chairman reported that the earlier recommendation of this committee that the Arizona State law regarding obligatory reporting of impaired physicians was discussed by the Association's Grievance Committee and the Board of Directors. At this point in time it has been determined that no attempt be made to amend that law.

Insurance Coverage

The chairman reported that the Medical Economics Committee of the Association was still considering the recommendations of the Physician Rehabilitation Committee relating to mandating insurance coverage for those persons suffering from alcoholism and drug abuse, and the matter still under review.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held in Pointe of View "C", The Pointe Resort, 7677 N. 16th Street, Phoenix, Arizona on Friday, April 25, 1980, a quorum being present, convened at 12:14 p.m., John T. Clymer, M.D., President and Chairman, presiding.

Committee Confirmations

Note: The single asterisk indicates reappointment. the double asterisk indicates new appointments. Those without asterisks are previous year appointments whose terms are still in effect. They are listed here for information only.

Articles of Incorporation and Bylaws Committee - Term - 1980-81

*Charles E. Henderson, M.D.
Chairman
Earl J. Baker, M.D., Secretary
John T. Clymer, M.D.
President
**Richard L. Collins, M.D.
*Philip E. Dew, M.D.
*Paul B. Jarrett, M.D.
*John K. Kerr, M.D.
Clyde W. Kurtz, M.D.
President-Elect
*W. David Rummel, M.D.
*Edward Sattenspiel, M.D.
*William C. Scott, M.D.
*Neil O. Ward, M.D.
*Willard F. Worthen, M.D.

Benevolent & Loan Fund Committee

	Term
*Arthur V. Dudley, Jr., M.D.	1979-82
Chairman	1980-81
Earl J. Baker, M.D.	1980-81
Secretary	
John T. Clymer, M.D.	1980-81
President	
Daniel C. DeBoer, M.D.	1979-82
Andrew M. Goldner, Ph.D.	1979-82
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
Pierre N. Levin, M.D.	1978-81
George S. Naifeh, Jr. M.D.	1978-81

Neopito L. Robles, M.D. 1980-81
Treasurer-Specified
Cecil C. Vaughn, Jr., M.D. 1979-82

Finance Committee

Neopito L. Robles, M.D. 1980-81
Chairman
Earl J. Baker, M.D. 1980-81
Secretary
John T. Clymer, M.D. 1980-81
President
Herbert G. Erhart, Jr., M.D. 1978-81
James L. Grobe, M.D. 1978-81
*Jerry W. Haggard, M.D. 1980-83
*John W. Kennedy, M.D. 1980-83
Clyde W. Kurtz, M.D. 1980-81
President-Elect
**John W. Magee, Jr., M.D. 1980-83
*C. T. Maloney, M.D. 1980-83
Theodore L. Mobley, M.D. 1979-82
**John B. Sawyer, M.D. 1980-83
*Seymour I. Shapiro, M.D. 1980-83
Maynard S. Smith, M.D. 1979-82
Harold C. Tretbar, M.D. 1978-81

Governmental Services Committee

*Louis C. Kossuth, M.D. 1980-83
Chairman 1980-81
Earl J. Baker, M.D. 1980-81
Secretary
John T. Clymer, M.D. 1980-81
President
John A. Davis, M.D. 1979-82
Charles Kalil, M.D. 1978-81
Clyde W. Kurtz, M.D. 1980-81
President-Elect
Wallace A. Reed, M.D. 1979-82
Robert St. John, M.D. 1979-82
*Marvin C. Schneider, M.D. 1980-83
**Bruce H. Shelton, M.D. 1980-83
Del Worthington 1979-82
Medical Student
Leon D. Zeitzer, M.D. 1978-81

Grievance Committee

William E. Crisp, M.D. 1980-81
Chairman
Richard W. Abbuhl, M.D. 1979-82
Earl J. Baker, M.D. 1980-81
Secretary
John T. Clymer, M.D. 1980-81
President
Philip E. Dew, M.D. 1978-81
Morton H. Dubnow, M.D. 1978-81
Arnold H. Dysterheft, M.D. 1978-81
*David E. Glow, M.D. 1980-83
John B. Jamison, M.D. 1978-81
Clyde W. Kurtz, M.D. 1980-81
President-Elect
*James N. Lane, M.D. 1980-83
R. T. McDonald, M.D. 1979-82
*Albert J. Ochsner, M.D. 1980-83

Health Manpower Committee

	Term
*Laurence M. Linker, M.D.	1979-82
Chairman	1980-81
Earl J. Baker, M.D.	1980-81
Secretary	
John T. Clymer, M.D.	1980-81
President	
*Brunce N. Curtis, M.D.	1980-83
Jospeh A. Dupont, M.D.	1979-82
Thomas H. Hicks, M.D.	1978-81
Louis C. Kossuth, M.D.	1978-81
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
**C. T. Maloney, M.D.	1980-83
Marcus M. Marble, M.D.	1979-82
**Robert R. Maxwell, M.D.	1980-83
*Stuart L. Posner, M.D.	1980-83
**Sara S. Reynolds, M.D.	1980-83
*Melvin I. Roth, M.D.	1980-83
George B. Rowland, M.D.	1978-81
Ronald P. Spark, M.D.	1978-81
Nettie White	1979-82
Medical Student	

History and Obituaries Committee

Marshall B. Block, M.D. 1980-81
Chairman
Earl J. Baker, M.D. 1980-81
Secretary
John T. Clymer, M.D. 1980-81
President
John R. Harlan, M.D. 1978-81
*John W. Kennedy, M.D. 1980-83
Clyde W. Kurtz, M.D. 1980-81
President-Elect
*W. David Rummel, M.D. 1980-83
*William C. Weese, M.D. 1980-83

Legislative Committee

*Richard O. Flynn, M.D. 1978-81
Chairman 1980-81
Earl J. Baker, M.D. 1980-81
Secretary
Wayne E. Beck, M.D. 1979-82
Daniel Bright, M.D. 1979-82
*Roy O. Bubeck, D.O. 1980-83
**William E. Cain, M.D. 1980-83
W. S. Chisholm, Jr., M.D. 1979-82
John T. Clymer, M.D. 1980-81
President
**William E. Crisp, M.D. 1980-83
Robert T. Daehler, M.D. 1980-83
**Richard Dale, M.D. 1980-83
William J. Dooley, M.D. 1979-82
Kenneth A. Dregseth, M.D. 1978-81
T. F. Griffin, Jr., M.D. 1978-81
Scott Hitt 1979-82
Medical Student
Clyde W. Kurtz, M.D. 1980-81
President-Elect
George Lastnick, M.D. 1978-81
Maureen Mangotich 1979-82
Medical Student
Richard McGill, D.O. 1978-81
Joseph B. McNally, M.D. 1979-82
*Thomas F. Moore, M.D. 1978-81
David A. Ogden, M.D. 1978-81
R. Michael O'Harra, M.D. 1979-82
*Kent L. Pomeroy, M.D. 1980-83
**Frederic L. Ruskin, M.D. 1980-83
Robert D. Sanford, M.D. 1979-82
*Edward Sattenspiel, M.D. 1980-83
J. L. Schamadan, M.D. 1979-82
Burt S. Strug, M.D. 1978-81
**Glen L. Sublette, M.D. 1980-83
William A. Susong, M.D. 1979-82
John W. Vosskuhler, M.D. 1978-81

Maternal and Child Health Care Committee

*William C. Scott, M.D. 1979-82
Chairman 1980-81
Earl J. Baker, M.D. 1980-81
Secretary
Frederic W. Baum, M.D. 1978-81
James F. Blute, III, M.D. 1979-82
Grace L. Busch, M.D. 1978-81
John T. Clymer, M.D. 1980-81
President
*Owen L. Cranmer, M.D. 1980-83
*William J.R. Daily, M.D. 1980-83
Jack H. Demlow, M.D. 1978-81
W. Droegemueller, M.D. 1979-82
**G. Wayne Figgs, M.D. 1980-83
Harold D. Gabel, M.D. 1980-83
John M. Gillette, M.D. 1978-81
*Raymond J. Jennett, M.D. 1980-81
*John V. Kelly, M.D. 1980-83
Susan Kower 1978-81
Medical Student
Clyde W. Kurtz, M.D. 1980-81
President-Elect
Walter K. Lippard, M.D. 1978-81
Gerald T. McMahon, M.D. 1978-81
H. Belton P. Meyer, M.D. 1979-82
Sister Alice Montgomery 1979-82
William J. Moore, M.D. 1979-82
William G. Payne, M.D. 1979-82
Hermann S. Rhu, Jr., M.D. 1978-81
**George H. Schade, M.D. 1980-83
Lewis S. Shenker, M.D. 1979-82
James M. Shwayder, M.D. 1978-81
William A. Susong, M.D. 1979-82
Paul A. Whitmore, D.O. 1979-82
Jack H. Wilson, M.D. 1979-82
Del Worthington 1979-82
Medical Student

Medical Economics Committee

*Clyde W. Kurtz, M.D. 1979-82
Chairman 1980-81
Salvatore D. Abate, M.D. 1979-82
R. S. Armstrong, M.D. 1978-81
Earl J. Baker, M.D. 1980-81
Secretary
Donald Buckly 1979-82
Medical Student
**James R. Callison, M.D. 1980-83
**William G. Chaffee, M.D. 1980-83
John T. Clymer, M.D. 1980-81
President
Charles F. Dalton, M.D. 1979-82
William M. Davis, M.D. 1978-81
Andrew Epstein 1979-82
Medical Student
James L. Frey, M.D. 1979-82
*David B. Gurland, M.D. 1980-83
**Lucy S. Hernried, M.D. 1980-83
A.D. Jacobson, M.D. 1978-81
Frederick W. Jensen, M.D. 1978-81
John F. Kahle, M.D. 1978-81

Laurence M. Linkner, M.D. 1978-81
Sheldon Marks 1979-82
Medical Student
**Theodore L. Mobley, M.D. 1980-83
Robert W. Weber, M.D. 1978-81
William R. Womack, M.D. 1979-82
R. Jack M. Zeluff, M.D. 1978-81

Medical Education Committee

	Term
*Robert E.T. Stark, M.D.	1978-81
Chairman	1980-81
Leon A. Asadourian, M.D.	1979-82
Earl J. Baker, M.D.	1980-81
Secretary	
James E. Brady, Jr., M.D.	1978-81
John T. Clymer, M.D.	1980-81
President	
Kenneth A. Dregseth, M.D.	1979-82
Palmer C. Evans, M.D.	1979-82
*Willard S. Gold, M.D.	1980-83
Samuel Goldfein, M.D.	1978-81
Stuart M. Gould, Jr., M.D.	1978-81
Harry W. Hale, Jr., M.D.	1979-82
Robert E. Hastings, M.D.	1979-82
Robert W. Hellmers, M.D.	1978-81
H. Peter Hernried, M.D.	1979-82
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
*Jack M. Layton, M.D.	1980-83
*Donald J. Logue	1980-83
Administrator	
James R. Mouer, M.D.	1979-82
Herbert N. Munhall, M.D.	1979-82
Alex Newman, M.D.	1979- 82
Eric G. Ramsay, M.D.	1979-82
Sharon Sandell	1979-82
Medical Student	
Julius Schwimmer, M.D.	1978- 81
Arnold L. Serbin, M.D.	1979-82
John J. Standifer, M.D.	1979-82
Lawrence Z. Stern, M.D.	1978-81
Ashton B. Taylor, M.D.	1978-81
*Charles A. Trahern, M.D.	1980-83
**Bryan R. Updegraff, M.D.	1980-83
Thomas E. Vorpahl, M.D.	1980-83
Anthony F. Vuturo, M.D.	1978-81
Albert G. Wagner, M.D.	1978-81
Walter S. Williams, M.D.	1978-81
Alan C. Winfield, M.D.	1979-82

Occupational Health Committee

*Joseph M. Hughes, M.D.	1980-83
Chairman	1980-81
Earl J. Baker, M.D.	1980-81
Secretary	
Floyd K. Berk, M.D.	1978-81
Earl M. Best, M.D.	1978-81
John T. Clymer, M.D.	1980-81
President	
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
Robert L. Levitin, M.D.	1979-82
J. L. Schamadan, M.D.	1979-82
E. W. Whiteford, Jr., M.D.	1979-82

Physician Rehabilitation Committee

Earl J. Baker, M.D. 1980-81
Paul B. Borgesen, M.D. 1978-81
Harrison G. Butler, M.D. 1979-82
John T. Clymer, M.D. 1980-81
President
Everett W. Czerny, M.D. 1979-82
*Howard S. Gray, M.D. 1980-83
**Wesley E. Green, M.D. 1980-83
**Lana L. Holstein, M.D. 1980-83
**Howard W. Kimball, M.D. 1980-82
Clyde W. Kurtz, M.D. 1980-81
President-Elect
*B. Ross Landess, M.D. 1980-83
Laurence M. Linkner, M.D. 1978-81
*Edward Meshorer, M.D. 1980-83
**Patrick T. Pphalen, M.D. 1980-83
**Sara S. Reynolds, M.D. 1980-83
Stephen C. Scheiber, M.D. 1979-82
Walter B. Tomlinson, M.D. 1979-82
*Mark A. Wellek, M.D. 1980-83
*Lincoln H. Westman, M.D. 1980-83
*Victor J. Zannis, M.D. 1980-83

Professional Committee

*Joseph W. Hanss, Jr., M.D. 1978-81
Chairman 1980-81
Earl J. Baker, M.D. 1980-81
Secretary
William C. Brainard, M.D. 1978-81
John T. Clymer, M.D. 1980-81
President
**M. M. DeArmond, M.D. 1980-83
Joseph A. Dupont, M.D. 1978-81
*Robert L. Hagan, M.D. 1980-83
Donald B. Kunkel, M.D. 1979-82
Clyde W. Kurtz, M.D. 1980-81
President-Elect
Clarence R. Laing, M.D. 1978-81
James G. Lawson, M.D. 1979-82
Sheldon Marks 1979-82
Medical Student
Warren L. Niccum, M.D. 1978-81
**Howard J. Reuben, M.D. 1980-83
**Tom D. Powell, M.D. 1980-83
Charles W. Pullen, M.D. 1978-81
James B. Scott, M.D. 1979-82
William J. Thomas, M.D. 1978-81
*W. R. VanNostrand, M.D. 1980-83
Del Worthington 1979-82
Medical Student

Public Relations Committee

**John T. Clymer, M.D. 1980-83
President & Chairman 1980-81
Earl J. Baker, M.D. 1980-81
*Marshall B. Block, M.D. 1980-83
Ronald L. Christ, M.D. 1978-81
Mrs. John T. Clymer 1980-83
Julian DeVries 1978-81
Medical Editor
Ira B. Ehrlich, M.D. 1979-82
Alan L. Gordon, M.D. 1978-81
Scott Hitt 1979-82
Medical Student
**William E. Ingram, M.D. 1980-83
Clyde W. Kurtz, M.D. 1980-81
President-Elect
Keith E. McReynolds, M.D. 1978-81
Stephen R. Stein, M.D. 1979-82
Diana Stockton 1979-82
Medical Student

Publishing Committee

	Term
*Marshall B. Block, M.D.	1980-81
Earl J. Baker, M.D.	1980-81
Secretary	
Burnell R. Brown, M.D.	1978-81
John T. Clymer, M.D.	1980-81
President	
C. Herbert Fredell, M.D.	1978-81
Benjamin K. Harris, M.D.	1979-82
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
**Jonathan M. Levy, M.D.	1980-83
William B. McGrath, M.D.	1978-81

Scientific Assembly Committee

*Suresh C. Anand, M.D.	1979-82
Chairman	1980-81
Earl J. Baker, M.D.	1980-81
Secretary	
*Floyd K. Berk, M.D.	1980-83
William W. Bohnert, M.D.	1978-81
George E. Burdick, M.D.	1979-82
W.S. Chisholm, Jr., M.D.	1979-82
John T. Clymer, M.D.	1980-81
President	
**Joel G. Cohen, M.D.	1980-83
Boyden L. Crouch, M.D.	1979-82
Joseph A. Dupont, M.D.	1979-82
T. R. Harrington, M.D.	1978-81
Thomas F. Hartley, M.D.	1978-81
Stuart I. Holtzman, M.D.	1979-82
*James M. Hurley, M.D.	1980-83
Clyde W. Kurtz, M.D.	1980-81
President-Elect	
*Philip Levy, M.D.	1980-83
**Luiz S. Lisboa, M.D.	1980-83
John E. Oakley, M.D.	1980-81
Vice President-Specified	
Peter Mao, M.D.	1979-82
J. Daniel Morris, M.D.	1979-82
*M. Herbert Nathan, M.D.	1980-81
William S. Nevin, M.D.	1978-81
Alex Newman, M.D.	1978-81
Charlotte Richards	1978-81
Medical Student	
*Neopito L. Robles, M.D.	1980-83
Marvin C. Schneider, M.D.	1979-82
Richard A. Silver, M.D.	1979-82
Donald P. Speer, M.D.	1978-81
*Lawrence Z. Stern, M.D.	1980-83
*Luis S. Tan, M.D.	1980-83
J. H. Targovnik, M.D.	1978-81
*Wilber C. Voss, M.D.	1980-83
*Donald J. Ziehm, M.D.	1980-83

Other Business

Ad Hoc Membership Committee

It was noted by the board that the Ad Hoc Membership Committee had been disbanded.

Annual Meeting Highlights

Presidential Address

John T. Clymer, M.D.

Delivered to the House of Delegates of the Arizona Medical Association on Wednesday, April 23, 1980, at 10:00 a.m.

This is a Decade for Decisions. I want to mention some of the changes that have had a major impact on how we practice medicine. Then, I want to tell you what two experts predict will be the issues organized medicine must face for this coming decade. Third, I will list those steps ArMA has taken to lead us into the 80's and finally, I will tell you what I think ArMA should do as a next step in becoming a stronger and more effective voice of Arizona physicians.

Changes in Medicine

My thoughts immediately go back to that hot Sunday afternoon July of in 1953 when I arrived in Phoenix. I was fresh out of an internship at the Henry Ford Hospital and was starting a general practice residency at the Maricopa County General Hosptial, then located at 35th Avenue and Durango.

My monthly stipend was $300.00 a month and the working conditions and the amount of time off was outlined by the hospital. Yes, I was part of the Housestaff, but there was no union representative to negotiate for me such items as salary, time off, and other working conditions. There was no PSRO and no Medical Audit. The closest that we came to that was the Tumor Board that met regularly and was chaired by Dr. Al Brewer. Ed Sattenspiel, Dick McMillan, and Dick Flynn will remember those times since they arrived on the scene at about the same time I did.

When I opened by own private office a year later in Phoenix, I learned the business aspects of running a medical practice the hard way. There were no Practice Managment Seminars, no cassette tapes, and few business consultants. We read *Medical Economics* for advice, and when we were really in a bind we went to one of the older docs for help and support. To mention a few there were Monroe Green, Bob McCulley, Charlie Henderson and Bill McGrath.

ArMA and the County Medical Society had no worries about membership then. You had to belong to get on a hospital staff. You had to belong to get malpractice insurance. Everybody that was anybody belonged to the medical society then.

Later, when the county hospital wanted to expand and move to a new location there was no need for a certificate of need. The greatest argument centered around where it should be located. The Federal Government encouraged hospital expansion through Hill-Burton funds.

Lucheon clubs for doctors serving education and social purposes were popular in Phoenix. The Gold-Headed Cane Club was full so we organized a new one: The Physicians Forum. Speakers at these noontime meetings kept us abreast of medically related topics. Warner Watkins was one of these speakers and represented an even earlier era in the practice of medicine. The scaley skin of his right hand with multiple small cancers and missing digits marked him as a first generation radiologist. We attended the meetings faithfully, but there was no need to sign in on those yellow cards for formal CME credit.

At that time the hospitals were the doctors' workshops. Who could conceive then—that by 1980—podiatrists, psychologists, chiropractors, and others would demand hospital privileges and access to diagnostic facilities.

Doctors had helped organize Blue Cross and Blue Shield to help patients pay the cost of medical care. They sat on the boards of the Blues, for their medical expertise was considered valuable. Now, it is a conflict of interest!

Yes, we all agree that the practice of medicine has changed. As Dorothy said to her faithful dog, Toto, in the "Wizard of OZ," "Toto, I don't think we're in Kansas any longer."

Issues Confronting Medicine

What are the issues that medicine must face for this decade. Listen to the predictions of two experts.

Attorney Betty Jane Anderson, an AMA counselor, sees continued pressure from the Federal Trade Commission. "B.J." as she is known,is one of the nation's foremost experts on health law. In a recent interview she said that the FTC is now interpreting health care as an industry, physicians and allied health professionals as tradesmen, and the interaction of hospitals, medical schools, the Blues, dental associations, medical societies and specialty associations as parts of a cartel!

The FTC and the Justice Department are focusing their attack on what they see as anticompetitive practices in the way health care is delivered, she continues. She is also concerned about the FTC establishing jurisdiction over the so-called health industry. Once you call a profession an industry, B.J. warns, you recognize government's affinity to regulate industry.

B.J. foresees closer scrutiny of JCAH standards by the FTC. Antitrust action on behalf of limited licensed practitioners seeking hospital privileges has already begun. Investigation of Board Certification requirements and exclusive medical care jurisdictions carved out by various medical specialties are not far off.

Dr. James Sammons, AMA Executive Vice-President, makes the following predictions for the 1980's. He reports that two out of three physicians will be either hospital-based, salaried, or in group practices. He says that medical associations will have to be advocates for these doctors as well as for the doctors in private practice. Medical associations will have to help hospital-based physicians deal with the institutions that employ them. Medical associations will have to assist physicians employed by the HMO's and other forms of health care delivery systems that may be controlled by non-physicians. Medical associations will have to offer consultive services to group practices. Medical societies and hospitals will have to join forces to combat unreasonable government regulations.

He continues by saying that jealousies between the town and gown must be set aside and all physicians join forces to fight sudden changes in federal policy that threaten the financial stability of our medical schools.

When Dr. Sammons was asked if the physician practicing quality medicine is an endangered species he replied, "Not just the quality physician is endangered, but so is the quality hospital." The public demands more and improved services but government cost restraints are leading toward rationing of medical services.

How true this is. . .and we're talking about government at all levels. When I read Dr. Sammons' statements, immediately an example came to mind right here in Tucson.

The student health service at the University of Arizona worked hard to win accreditation as an ambulatory care unit by the JCAH. They gained that accreditation a year ago. It indicated they provided high quality care in a first class physical plant. Yet, Dr. DeArmond, the director of the student health services told me he has been under extreme political pressure from the legislature to reduce expenditures. How can he do that without reducing services in some fashion?

Returning to Dr. Sammons' predictions, he sees a big drive for physicians' assistants, nurse extenders, nurse practitioners and others to be freestanding and directly reimbursed under government programs. As a result, he sees the emergence of a separate, competitive system of non-physician providers.

ArMA Takes Action

What about PSRO's and health planning? Dr. Sammons says that health planning and PSRO's are the law of the land and he maintains that if the current programs were to be abolished they could be replaced by some thing worse! He concludes by saying, "We have to live with the reality of government encroachment."

What steps has ArMA already taken to lead us into the 1980's? The list is in your handbook. Those activities that particularly relate to my remarks today include continuing medical education, the 1980 Membership Development Plan, improving the doctor-patient relationship as recommended in his report by Dr. Stephen Brown, our activity in reviewing health legislation, position papers on various limited licensed practitioners, educating physicians in dealing with health planners, teaching physicians how to communicate medicine's story, and closer liaison with our auxiliary.

The last part of this talk is the question of where does ArMA go from here. I say **now** is the time that ArMA involve itself in a formal planning process. This process would include the gathering together of a representative group of ArMA members, lead by an expert in the field of planning. Through a representative poll, the opinions of the membership will be sought out. This group will attempt to define the present purpose of the organization, its resources and then finally, set forth two or three goals that the organization can reasonably expect to achieve within the next two to three years. Included in the process is the mechanism whereby the organization is kept ontrack toward the achievement of the goals.

What do I want from you? I want your active participation. Get actively involved in the committees that you are assigned to, become better informed on the issues that medicine faces today and be reasonable when you discuss your point of view with your patients, the general public and the decision makers. If we, the officers of ArMA, do something you like, tell us about it. If we do something you don't like, tell us about it; but, at the same time, make suggestions for improvement.

Ladies and Gentlemen, this is our Decade for Decision.



ArMA Officers, Speaker of House and Parliamentarian start the House of Delegates proceedings.



Hoyt D. Gardner, M.D., President - AMA



Mrs. Eloise Clymer
Past-President, ArMA Auxiliary



Jack M. Layton, M.D. makes a point



President Clymer ponders a point



Mrs. Harry S. Dvorsky
1st V.P. AMA Auxiliary



Daniel T. Cloud, M.D., AMA Trustee & Candidate for the Presidency of the AMA



Newly elected Vice President
John E. Oakley, M.D.



James M. Hurley, M.D. ready to address the House



The House of Delegates in action



V-P, President-Elect, and President plan the future



Mrs. B.J. Anderson, Assistant General Counsel, AMA discusses program with Dr. Clymer and others



Mrs. Clymer and ArMA Auxiliary President, Mrs. Newman compare notes



President-Elect
Clyde W. Kurtz, M.D.



The President's table at the beautiful President's Banquet



President John T. Clymer, M.D.



Past President Wm. E. Crisp, M.D. receives the much coveted Past-Presidents plaque from Dr. Clymer



Mrs. Ronald Johnson presents AMA-ERF check to Dr. Kettel, Dean of the College of Medicine



ArMA's first annual Fun Run takes off -



Mrs. Richard L. Collins
ArMA Auxiliary



Carlos C. Craig, M.D. receives one of the eleven 50 Year Club Awards presented at the President's Banquet

The Arizona Medical Association, Inc. Resume

of the House of Delegates 1980 Annual Meeting April 25, 1980

Adjourned meeting of the House of Delegates of the Arizona Medical Association, Inc. held in the Grand Ballroom of the Pointe Resort, Phoenix, Arizona, Friday, April 25, 1980, convened at 8:07 a.m., Robert A. Price, M.D., Speaker of the House, presiding.

Credentials

The Credentials Committee reported a quorum present and the House duly constituted.

Roll Call

On the roll call 104 Delegates (and/or Alternates) and 27 members of the Board of Directors were present.

Minutes

Minutes of the meeting of the House of Delegates held April 27, 1979, were approved as distributed.

Election of Officers

The following officers were elected for a term of one year. The following were elected for a term January 1, 1981 to December 31, 1982.

President-Elect	Clyde W. Kurtz, M.D.
Vice President	John E. Oakley, M.D.
Secretary	Earl J. Baker, M.D.
Treasurer	Neopito L. Robles, M.D.
Speaker of the House	Robert A. Price, M.D.
Editor-In-Chief	Marshall B. Block, M.D.

Election of District Directors

The following District Directors were elected.

Central District Dir.	R. L. Collins, M.D.	(1980-83)
Central District Dir.	H. S. Gray, M.D.	(1980-83)
Central District Dir.	R. Kirschner, M.D.	(1980-83)
Northwestern District Director	R. Gardner, M.D.	(1980-82)
Southeastern District Director	K. A. Dregseth, M.D.	(1980-83)
Southern District Director	C. T. Maloney, M.D.	(1980-83)
Southern District Director	M. Fuchs, M.D.	(1980-83)
Southwestern District Director	J. F. Martin, M.D.	(1980-83)

Reference Committee on Resolutions

Report of the Reference Committee on Resolutions as deleted, amended or as other wise disposed of, adopted with the following actions taken on motions regularly made and carried.

Resolution 1-80

Subject: Unified membership between the County Medical Societies and the Arizona Medical Association.

Whereas, The Board of Directors has introduced a resolution proposing a bylaw amendment which, if adopted would eliminate State-National unified membership by removing the requirement of membership in the American Medical Association as a condition for membership in the Arizona Medical Association; and

Whereas, Interest has been expressed in additionally eliminating County-State unified membership by removing the requirement of membership in the Arizona Medical Association as a condition for membership in a County Medical Society; and

Whereas, It is recognized that eliminating County-State unified membership requires a more extensive amendment of the bylaws, involving policy decisions, the results of which will affect the method of operation of both the State and County Societies and involves different factors relating to the overall desirability of making such a change from those relating to State-National unification; now, therefore, be it

Resolved, That the President appoint a Committee to determine the required bylaw changes, including the policy decisions needed therefor, evaluate the operational impact of these changes on the State and County Societies and determine the desirability of eliminating County-State unified membership; and, be it further

Resolved, That the above-mentioned Committee report its findings and recommendations to this House of Delegates at its 1981 meeting.

Adopted, 4/25/80

Resolution 2-80

Subject: 1981 Calendar Year Budget of Income and Expense

Whereas, It is customary for the House of Delegates to approve the Budget of Income and Expenditures for the next succeeding calendar year of the Arizona Medical Association, Inc.; therefore, be it

Resolved, That the following Budget of Income and expenditures for the calendar year 1981 be adopted: ;and be it further

	Income	Expenditures	
General Depart.	$821,915.00	$717,005.00	$104,910.00
Committee Depart.	13,500.00	69,000.00	(55,500.00)
Annual Meeting Department	59,200.00	55,590.00	3,610.00
Publishing Depart.	99,350.00	97,660.00	1,690.00
Total	$993,965.00	$939,255.00	$ 54,710.00

Resolved, That the annual dues of the Arizona Medical Association, Inc. be $300 for Active members, with $280 going to General Department Budget and $20 going to the Publishing Department Budget; and be it further

Resolved, That the Service member dues be $75, with $55 going to the General Department Budget and $20 going to the Publishing Department Budget; and be it further

Resolved, That the Intern or Resident Active member dues be $30 with $10 going to the General Department Budget and $20 going to the Publishing Department Budget, and be it further

Resolved, That the Intern or resident Active member dues be waived for the first year of Internship or Residency.

Adopted, As Amended, 4/25/80

Resolution 3-80
Subject: Statutory Definition of Death - American Medical Association Model Legislation

Tabled, 4/25/80

Resolution 4-80
Subject: American Association of Medical Assistants (A.A.M.A.)

Whereas, The American Association of Medical Assistants, Arizona Society, is a nonprofit organization with five chapters in Arizona, and

Whereas, The objectives of this association are to inspire *members to render honest, loyal, more efficient service to the profession and the public; to strive to cooperate with the profession in improving public relations; to render educational services for the self-improvement of its members and to stimulate a feeling of fellowship and cooperation among the societies; and to encourage and assist in forming local and state assistants societies, and

Whereas, This association provides certification programs in basic (CMA), administrative (CMA-A) and clinical (CMA-C) administered by the National Board of Medical Examiners; certification revalidation; Guided Study, tri-level continuing education with C.E.U. credit; a professional journal, state and chapter publications; Maxine Williams Scholarships nationally, Karl Witte Memorial scholarships statewide, and

Whereas, AAMA has accredited over 135 medical assisting programs nationally through curriculum review in collaboration with the Committee on Allied Health Education and Accreditation (CAHEA), an independent agency sponsored by the AMA, and

Whereas, CAHEA/AAMA has been recognized as an official accrediting agency for medical assisting programs by the U.S. Office of Education and the Council on Post-Secondary Accreditation, and

Whereas, The AMA House of Delegates, on five occasions, urged every physician to become fully acquainted with the objectives and programs of the AAMA and encourage membership at the same time providing over 2000 AMA members to serve as advisors to AAMA, therefore be it

Resolved, That the members of the Arizona Medical Association compliment the AAMA, Arizona Society leadership, and encourage their employees to become AAMA members and participate in their educational, certification and chapter programs for improved effective medical service.

*Medical assistants are allied health practitioners who work in doctors' offices, hosptials and medical facilities, performing administrative and/or clinical duties under the direct supervision of a physician.

Adopted, 4/25/80

Resolution 5-80
Subject: Principles of Medical Ethics

Whereas, After careful review of the various proposals for revision of the Principles of Medical Ethics, the Board of Directors of the Arizona Medical Association feels that the present Principles continue to be adequate, with some minor revisions, therefore be it

Resolved, that the Association support the continuation of the Principles of Medical Ethics as revised in 1957 and presently in existence, with the following modifications: 1) The deletion of the second portion of Section 3 thereof; 2) the deletion of the last sentence of Section 5 thereof; 3) the deletion of the words "and the patient's ability to pay" in Section 7 thereof; and 4) the addition of a Section 11, setting forth in essence, the following language: "Physicians shall not solicit patients, nor shall physicians advertise in a false, fraudulent, deceptive or misleading manner or advertise the quality of medical service," the edited version to read as follows:

Preamble. - These principles are intended to aid physicians individually and collectively in maintaining a high level of ethical conduct. They are not laws but standards by which physicians may determine the propriety of their conduct in relationship with patients, with colleagues, with members of allied professions, and with the public.

Section 1. - The principle objective of the medical profession is to render service to humanity with full respect for the dignity of man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion.

Section 2. - Physicians should strive continually to improve medical knowledge and skill, and should make available to their patients and colleagues the benefits of their professional attainments.

Section 3 - Physicians should practice a method of healing founded on a scientific basis.

Section 4 - The medical profession should safeguard the public and itself against physicians deficient in moral character or professional competence. Physicians should observe all laws, uphold the dignity and honor of the profession and accept its self-imposed disciplines. They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession.

Section 5. - Physicians may choose whom they will serve. In an emergency, however, they should render service to the

best of their ability. Having undertaken the care of patients, they may not neglect them; and unless they have been discharged they may discontinue their services only after giving adequate notice.

Section 6. - Physicians should not dispose of their services under terms or conditions which tend to interfere with or impair the free and complete exercise of medical judgement and skill or tend to cause a deterioration of the quality of medical care.

Section 7. - In the practice of medicine, physicians should limit the source of their professional income to medical services actually rendered by them, or under their supervision, to their patients. Fees should be commensurate with the services rendered. Physicians should neither pay nor receive a commission for referral of patients. Drugs, remedies or appliances may be dispensed or supplied by physicians provided it is in the best interests of the patient.

Section 9. - Physicians may not reveal the confidence entrusted to them in the course of medical attendance, or the deficiencies they may observe in the character of patients, unless required to do so by law or unless it becomes necessary in order to protect the welfare of the individual or of the community.

Section 10. - The honored ideals of the medical profession imply that the responsibilities of physicians extend not only to individuals but also to the society where these responsibilities deserve their interest and participation in activities which have the purpose of improving both the health and the well-being of the individual and the community.

Section 11. - A physician shall not engage in in-person solicitation of patients, nor shall physicians advertise in a false, fradulent, deceptive or misleading manner or advertise the quality of medical service.

; and be it further

Resolved That the Arizona delegates to the American Medical Association be instructed to submit this Resolution to the AMA House of Delegates for its consideration during the 1980 Annual Meeting.

Substitute Resolution adopted, as Amended, 4/25/80

Reference Committee on Amendments

Report of the Reference Committee on Amendments as deleted, amended or as otherwise disposed of, adopted with the following actions taken on motions regularly made and carried.

(Note: All bracketed, italicized words represent deletions; all fully capitalized words are new material.)

Resolution A-1-80

Subject: Unified Membership - American Medical Association

Not Adopted, 4/25/80

Meeting adjourned Sine Die at 11:28 a.m.

Future Medical Meetings

The following institutions and organizations hav been accredited for their continuing medica education programs by the Liaison Committee o Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

The Third International Conference on the Adjuvant Therapy of Cancer will be held a the Tucson Convention Center, Wednesday March 18 through Saturday, March 21, 1981 under the sponsorship of the Cancer Center Division of the University of Arizona. Drs Sydney E. Salmon and Stephen E. Jones wil serve as Conference Co-Chairmen. Abstrac forms are now available; the deadline for submisison of abstracts (prepared in the format of AACR/ASCO) is November 1, 1980 For abstract forms or futher information, please contact: Mary Humphrey, Conference Coordinator, Cancer Center Division, University of Arizona, Tucson, Arizona 85724.

JULY

Advances in Therapeutics
July 11-12. Little America, Flagstaff, AZ. Sponsor & Contact: University of Arizona College of Medicine; Office of CME and Outreach, Tucson, AZ 85724. Approved for 10 hours of category 1 credit.

dvanced Cardiac Life Support Program Recertification)
uly 11. Arizona Medical Assn., 810 W. ethany Home Rd., Phoenix, AZ. Sponsor: .C.E.P., Arizona Affiliate-American Heart ssn. and ArMA. Contact: Carol Turner, rMA Offices. Approved for 8 hours of ategory 1 credit

ay of Surgery
uly 25. Little America, Flagstaff, AZ. ponsor: Arizona Chapter, American College f Surgeons. Contact: Stephen L. /angensteen, M.D., Chairman, Dept. of urgery U of A College of Medicine. Tucson, Z 85724. Approved for 5 hours of category 1 redit.

dvanced Cardiac Life Support Program Provider Course)
uly 25-26. Arizona Medical Assn., Phoenix, Z. Sponsor: A.C.E.P., Arizona Affiliate-merican Heart Association and ArMA. ontact: Carol Turner, ArMA Offices. pproved for 16 hours of category 1 credit.

oconino County Medical Society Summer ledical Seminar
uly 26-27. Little America Motor Inn, lagstaff, AZ. Sponsor: Arizona Medical ssociation and Coconino County Medical ociety. Contact: Thomas DaVee, M.D., 710 . Beaver, Flagstaff, AZ 86001. Approved for hours of category 1 credit.

AUGUST

rizona Perinatal Medicine Program
ugust 8 & 9, Yavapai Community College, rescott, Arizona. Sponsor: Arizona Perinatal rogram, 801 W. Bethany Home Rd., hoenix, AZ. Contact: Arizona Perinatal rogram Offices. Approved for 15 hours of ategory 1 credit.

ymposia de Santa Fe
ugust 21-23. Santa Fe Hilton, Santa Fe, New lexico. Sponsor: Symposia de Santa Fe. ontact: W. J. Levy, M.D., Sec.-Treasurer, .O. Box 5175, Coronado Station, Santa Fe, ew Mexico 87502.

SEPTEMBER

ersonal Instruction Course
Intra & Extra Capsular Cataract Extraction ith IOL Implantation.
eptember 10-13 and 24-27, Sponsor and ontact: The Eye Foundation, Ronald W. arnet, M.D., Medical Director, 421 N. 18th treet, Ste 105, Phoenix, AZ 85006.

omparative Techniques of Weight Control
eptember 26-27, Scottsdale Hilton Hotel, cottsdale, AZ Sponsor: University of rizona College of Medicine, and the Central rizona District Dietetic Association. ontact: Janet Price, R.D., CADDA, 1051 S. obson Rd., No. 56, Mesa, AZ 85202. pproved for 10 hours of category 1 creditl.

OCTOBER

ersonal Instruction Course
Intra & Extra Capsular Cataract Extraction ith IOL Implantation
ctober 8-11 and 22-25. Sponsor and ontact: The Eye Foundation, Ronald W. arnet, M.D., Medical Director, 421 N. 18th treet, Ste 105, Phoenix, AZ 85006.

NOVEMBER

Personal Instruction Course
In Intra & Extra Capsular Cataract Extraction with IOL Implantation
November 5-8 and 19-22. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 19th Street, Ste 105, Phoenix, AZ 85006.

Medical Society of the United States and Mexico
November 12-15, Guadalajara, Jalisco, Mexico. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Exec. Secy., 3161 N. Pantano Road, Tucson, AZ 85715.

DECEMBER

International Congress on Ericksonia Approaches to Hypnosis and Psychotherapy.
December 4-8, Phoenix, Arizona. Sponsor: Maricopa County General Hospital and The Milton H. Erickson Foundation, Inc. Contact: Jeffrey K. Zeig, Ph.D. The Milton H. Erickson Foundation, Inc. 1935 East Aurelius Avenue, Phoenix, Arizona 85020. Approved for hour per hour category 1 credit.

1981

FEBRUARY

Third Annual International Cataract Surgery Symposium
February 16-18, 1981. Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.
Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.
A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.
Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.
Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.
Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.
Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists
Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.
Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.
Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.
Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.
Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.
Tumor Board
last Thursday of the month, 12 Noon, Educ. Center Classroom I & II, May, July, Sept, & Nov.
Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.
Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.
Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.
Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

EL DORADO HOSPITAL TUCSON, (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.
Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.
Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 A.M., Contact: Paul Bergeson, M.D.
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM & R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.
Attending's Hour
5th Monday, 12:30 P.M., Contact: J. Goldberger, M.D.
Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman
Ophthalmology Conference
1st Monday, 12:30 P.M., Contact: Bruce Gillers, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.
Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: B. Phibb M.D.
Clinical Pharmacology Conference
5th Tuesday, 12:30 P.M., Contact: M. Kat Clinical Pharm.
12Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact Laguna, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: M. Bake M.D.
Medical Surgical Grand Rounds
2nd Wed., 12:30 P.M., Contact: Chief Medic Resident
Medical Conference
1st Wed, 12:30 P.M., Contact: Chief Medic Resident
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: B. Phibb M.D.
Endocrine Conference
2nd Thursday, 12:30 P.M., Contact Munjack, M.D.
GI Conference
4th Thursday, 12:30 P.M., Contact: C. Kanu M.D.
Mortality & Morbidity Conference
3rd. Thursday, 12:30 P.M., Contact: Munjack, M.D.
OB/GYN Dept. Meeting
2nd Thursday, 2 P.M., Contact J. Wurze M.D.
OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: Wurzel, M.D.
OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 P.M., Contact: J. Wurze M.D.
Clinical Pathology
5th Thursday, 12:30 P.M., Contact: Fleishman, M.D.
Renal Conference
1st Friday, 12:30 P.M., Contact: T. Penc M.D.
Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D
Rheumatology Conference
3rd Friday, 12:30 P.M., Cntact J. Goldberge M.D.
Chest Conference
5th Friday, 12:30 P.M., Contct: D. Engelsber M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 8500
Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 8500
Contact: Leonard Tamsky, M.D.
Medicine Conference
Daily 12-1 P.M., Contact: S. Schaffner, M.D
Chest Surgery Conference
Weekly, Mondays, 1:30 P.M., Santa Cruz Rm
Hepatology Conference
2nd Tuesday, 1:00 P.M., Contact: Wa Koppenbrink, M.D.
OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Roo
Obstetrical Problem Conference
Weekly Tuesday, 7:30 A.M., Yavapai Rm.

Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.,Contact: Robert Ganelin, M.D.
Urology Discharge Planning Conference
Weekly, 11:30 A.M., Station 42
Hand Surgery Conference
Weekly Wed, A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly, Wed. 7:30 A.M., Yavapai Rm.
Clinical Psychiatric Conference
Weekly, Wed., 11-12 P.M. Mental Health Annex, Rm. 1346.
Surgery Conference
Weekly Wed., 7-8 A.M., Surgical Dept.
Cardiology Conference
Weekly Thurs., 2 P.M., Santa Cruz Rm.
OB/GYN Resident Conference
Weekly Thurs., 12 P.M., Yavapai Rm.
GYN Endocrine Seminar
1st & 3rd Friday, 12:30 P.M., Sanata Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & HEALTH CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.
Medical & Surgical Conference
Weekly Monday, Wed. and Friday, 12 Noon
OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conf. Rm.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for Category 1 credit.
Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Contact: D. Randall, M.D.
Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 A.M.
Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 A.M., Conf. Rm. A, Contact: Drs. L. Moher & L. Fairbanks.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for Category 1 credit.
Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Clinical Conferences
Weekly Tuesday, 12 Noon, Kiva Conference Room
Tumor Board Conference
Weekly Friday, 12 P.M., Kiva Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.
Family Practice Conference
1st Monday, 12:30 P.M., Doctors' Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiolpulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs, 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical—Surgical Rdiological Conf.
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.
OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Genetics Conference
Weekly Monday, 12:30 P.M., Pediatric Department
Pediatric Rounds
Weekly Monday, Wed., & Friday, 10:30 A.M., Pediatric Department
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.
ECG. Conference
Weekly Tuesday, 12:30 P.M., Pediatric Department
Medical Grand Rounds
Weekly Wednesday, 8 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 8 A.M., PIMC
Visiting Professor Informal Rounds
Weekly Thursday, 9:30 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 12:30 P.M., PIMC.
Nephrology Conference
Weekly Friday, 12:30 P.M., Pediatric Department

ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N. Wilmot Road, Tucson, AZ. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Family Practice Department Meeting
3rd Tuesday, 12 P.M., Contact Wm. Moteforte
Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 P.M., Contact: Kim Sowards.
Current Concepts in Medicine
Weekly Tuesday, 12 P.M., Auditorium.
Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 P.M., Contact: S. Salmon, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Emergency Medicine Lectures
Weekly Thursday, 8 A.M., Century Room A
Mental Health Update
1st Friday, 11:30-1:00 P.M., Century Room A
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.
Cardiac Conference
Weekly Monday, 12:15 P.M., Auditorium
Surgery Conference
1st Tuesday, 12:15 P.M., Auditorium
Emergency Medicine Conference
1st Wednesday, 12:15 P.M., Auditorium
Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 P.M., Auditorium
Psychiatry Conference
3rd Thursday, 7 A.M., Auditorium
Combined Medical-General Practice Conference
1st Friday, 12:15 P.M., Auditorium
Toxicology Grand Rounds, Auditorium
1st Friday, 7:30 A.M., Auditorium
Ophthalmology Conference
1st Saturday, 8:30 A.M., Auditorium.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Cardiology Conference
Weekly Monday, except last, 12 Noon, Contact: J. Fernadez, M.D.
Dermatology Conference
4th Monday, 5:00 P.M., Contact: R. Miller, M.D.
Endocrinology Conference
Last Monday, 12 P.M., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.
Chest Conference
3rd Tuesday, 12 Noon, Contact: William Nevin, M.D.
Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Infectious Disease Conference
2nd Tuesday, 12 Noon, Contact: B. Friedman, M.D.

Nephrology Conference
1st Tuesday, 12 Noon, Contact: Dr. Seltzer.
Orthopedic Conference
1st Tuesday, 8:00 P.M., Contact: Jay Katz, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 P.M., Contact: Dr. Lightner.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.
Clinical Pathology Conference
Last Wednesday, 8:00 A.M., Contact: Dr. Fuchs.
Family Practice Meeting
2nd Wednesday, 12:30 P.M., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.
Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buschsbaum, M.D.
Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.
Tumor Conference
Weekly Thursday, 12 Noon, Contact: Dr. Hastings.
G—I Conference
Weekly Friday, 12 Noon, Contact: C. Krone, M.D.
Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.
OBGYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.
OBGYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.

Surgical Department Confernece
Weekly Saturday, 7:30 A.M., Contact: Dr. Hastings, TMC.
Surgical Grand Rounds
Weekly Saturday, 9:00 A.M., UAHSC-Room 5403, Contact: Dr. Norton.

PHOENIX VETERANS ADMINISTRATION MEDICAL CENTER

7th Street and Indian School Road, Phoenix, AZ 85012. Contact: Alfred Heibrumn, M.D. Approved for Category 1 credit.
GI X-Ray Clinical Correlation Conf.
1st & 3rd Monday, 1 P.M., Rm. C435, Contact: Dr. Sanowski, Ext 618.
Medical/Surgical GI Conference
1st & 3rd Monday, 3 P.M., Rm. 3134, Contact: Dr. Kozarek, Ext 413. Dr. Mertz, Ext 493.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 P.M., RM. 3134, Contact: Dr. Mertz, Ext 493.
GI Pathology Conf.
2nd & 4th Monday, 1 P.M., Path Conf. Rm, Contact: Dr. Sakowski.
Vascular Conference
2nd Monday, 3 P.M., Room 3134, Dr. Cintora, Ext 419
Surgery/Pathology Conference & Tissue Review
1st & 3rd Tuesday, 4 P.M., Room 2128, Contact: Dr. Landard
Surgery Mortality/Morbidity Conference
4th Tuesday, 4 P.M., Room 3134, Contact: Dr. G. Mertz.
GI Grand Rounds
Weekly Tuesday, 1 P.M., Contact: Drs. Sanowski, Kozarek & Partyka
Cancer Symposium
Weekly Wed., 1 P.M., T-5, Contact: Dr. Byrne, Ext. 426.
Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 P.M., Contact: Dr. Rohwedder, Ext. 388.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 P.M., Rm 3134. Contact: Dr. Mertz, Ext. 493.
Medical Surgical Chest Conference
1st & 3rd Thursday, Contact: Dr. Rohwedder or Dr. Gill.
Cardiology Conference
2nd Thursday, 1 P.M., Rm. T-5.
Medical Service Grand Rounds
Weekly Friday, 11 A.M., Contact: Dr. Zeller
Surgical Journal Review & Videotape Session
Weekly 1 P.M., Room 3134.

PRESCOTT VETERANS ADMINISTRATION HOSPTIAL MEDICAL CENTER

Prescott, Arizona 86313. Contacts listed below. Approved for category 1 credit.
Medical Lectures
1st & 2nd Thursday & Weekly,Fridays, 1:30 P.M., Bldg. 28. Contact: J. W. Fitzharris, D.O.
GI Rounds
Third Thursday, 10 A.M.-2:30 P.M., Contact: J. W. Fitzharris, D.O.
Medical Rounds
First & seond Thursday, 1:30 P.M., Bldg. 28, Contact: J. W. Fitzharris, D.O.

TUCSON

VETERANS ADMINISTRATION HOSPITAL & MEDICAL CENTER (U of A Tucson)

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.
Medical Surgical Chest Conference
Weekly Tuesday, 2 P.M., Contact: Dr. Young
Medical Grand Rounds
Weekly Wed., 12-1:00 P.M., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam
Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.
Grand Rounds
Weekly, Thursday, 11 A.M., Bldg. 107, Contact J. Fitzharris, D.O.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon.
Neurology Grand Rounds
Weekly Friday, 12 P.M., Contact: Dr. Sibley.

YUMA REGIONAL MEDICAL CENTER (U of A, Tucson/ArMA)

2400 Avenue A., Yuma Az 85364. Contact:Alan Winfield, M.D. Approved for category 1 credit.

Radiology Conference
1st Tuesday, 7:00 A.M.
Operation Outreach
2nd Tuesday, 6:30 P.M.
Pathology Conference
4th Tuesday, 7:00 A.M.
Operation Outreach
2nd Wednesday, 7:00 A.M.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.
Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact: Drs. Otto & Zehngut.
Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 P.M., HSC Auditorium, Contact: Cancer Center
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning

Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Room 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.

Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn

Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern

Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.

Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.

Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford

Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room

Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross

Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross

Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.

Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.

Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley

Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.

Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.

Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson

Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones

Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson

Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655

Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich

Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.

Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.

Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.

General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

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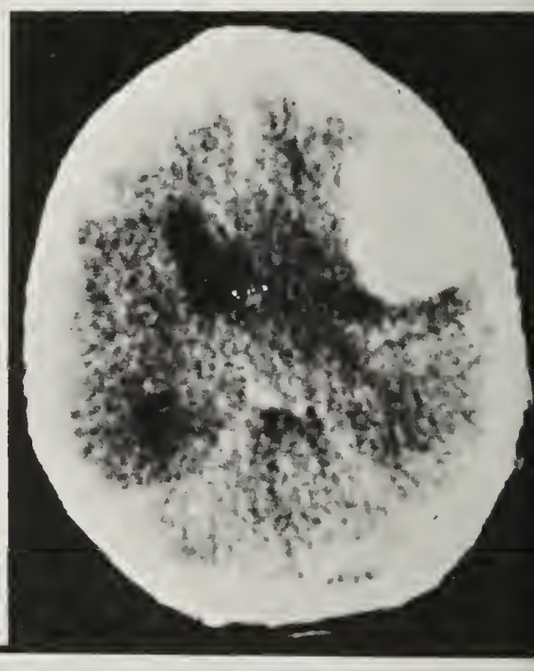
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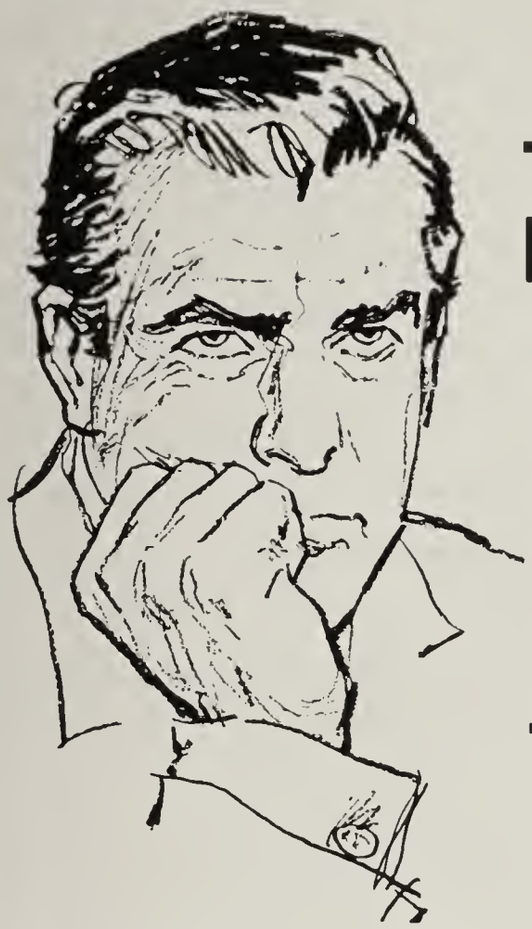
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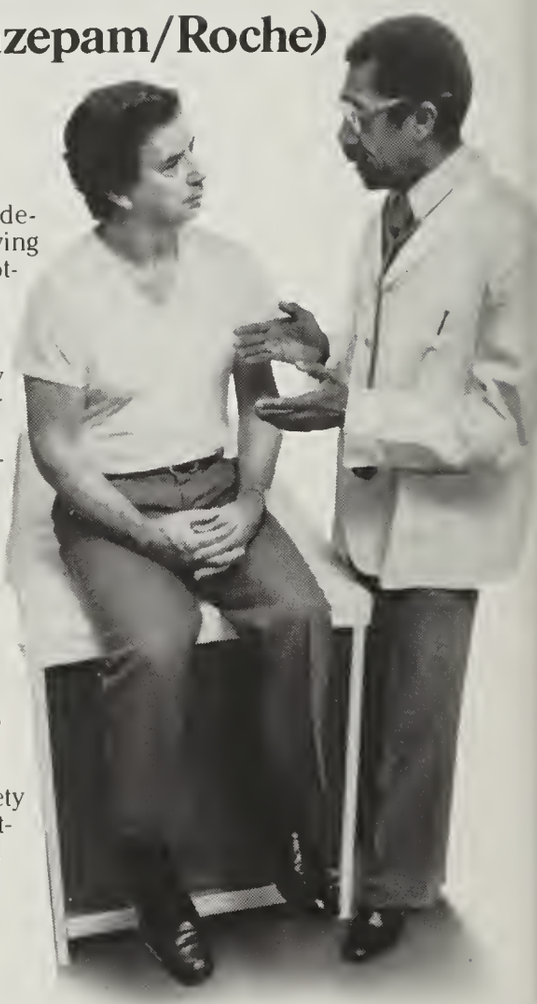
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Monitoring patient response to Valium® (diazepam/Roche)

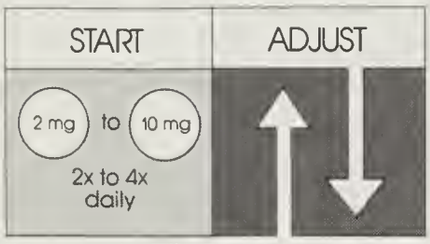


Assessing initial response to therapy

During the first follow-up visit after initiating therapy, both physician and patient should determine if Valium (diazepam/Roche) is having the desired effect. Most patients will promptly report a feeling of relaxation and relief of anxiety-linked symptoms such as insomnia, headaches, palpitations and hyperventilation. You will probably observe that the patient is calmer and more relaxed. If, however, patient response does not measure up to expectations, a reevaluation of the patient's profile with modification of the dosage regimen should be considered.



Making dosage adjustments



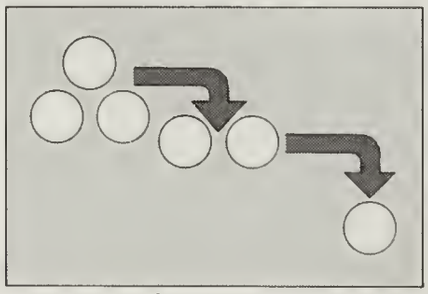
With any psychoactive medication it is good medical practice to initiate therapy at base dosage levels and titrate to the patient's needs. With Valium, experience has shown that 5 mg t.i.d. is usually sufficient although some patients with severe or persistent anxiety may require higher dosages initially. In geriatric or debilitated patients, the recommended dosage is 2 to 2½ mg once or twice daily. When anxiety fluctuates, as is common with most patients, the dosage may be adjusted as needed during the course of therapy; three strengths in scored tablets give you unmatched flexibility and simplicity in individualizing dosage.

Evaluating progress toward therapeutic goals

SET GOALS						
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

At the beginning of therapy it is now common practice for both physician and patient to establish treatment goals and to estimate the amount of time needed to achieve them. Then the patient knows what to expect and when to expect it. Some physicians find that compiling a checklist of presenting symptoms and complaints is useful for assessing the patient's response from visit to visit. In this way, progress toward attainment of the therapeutic goal is reviewed at regular intervals. As patients feel their symptoms abate and begin to develop insight into the sources of their anxiety and psychic tension, the checklist can be expected to dwindle.

Discontinuing pharmacologic intervention



When you decide to discontinue therapy, tapering dosage is good medical practice. Although rarely necessary after short-term treatment with Valium, gradual dosage reduction is advisable for patients who have been on extended therapy. This gradual discontinuance should preclude either recurrence of pretreatment symptoms or development of untoward side effects. Symptoms of withdrawal have almost always been associated with abrupt discontinuance of therapy at higher dosages taken continuously over long periods of time.

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Valium® (diazepam/Roche) ℞

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Indications: Tension and anxiety associated with anxiety disorders, transient situational disturbances and functional or organic disorders, psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy)

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses. Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months. After extended therapy, gradually taper dosage. Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d., alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d., adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500, Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10, Prescription Paks of 50, available in trays of 10.

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- Personal effectiveness training, which teaches the anorexic how to express ideas, thought and feelings... how to identify and manage stress.
- Art-Music-Movement therapies to help reconcile emotional conflicts and reduce the anorexic's distorted body image.
- Psychodrama, which helps the anorexic develop effective ways to communicate and gain a greater sense of emotional independence. Families are encouraged to participate in the psychodrama experience.

Hospitalization can be extremely helpful in breaking the binge eating-vomiting cycle that affect many anorexics. Camelback Hospitals offer a highly specialized approach to diet and nutrition, along with the services of a professional medical house staff ...on call 24 hours a day.

Other Services

In addition to the treatment program for anorexia nervosa, Camelback Hospitals offer

- Alcohol and Drug Abuse Programs
- Day Treatment Programs
- Young Adult Treatment Programs
- Adolescent Treatment Programs (patients 13 to 17 years of age participate in both the Adolescent and Anorexic Programs)
- Acute psychiatric care for adults
- Geriatric Treatment Program
- Family, group and individual psychotherapy
- Occupational and recreational therapy — Psychodrama
- Social Services

About the Hospitals

Camelback Hospitals, located in Phoenix and Scottsdale, Arizona, provide intensive, multidisciplinary diagnostic and short term treatment designed to alleviate psychiatric symptoms, increase self-awareness and build psychosocial competence.

Special emphasis is placed on sharing with each patient a more complete understanding of himself, so that he can participate in his therapy with insight and intelligence.

An open medical staff of more than 60 psychiatrists, psychologists, psychiatric nurses, social workers and activity therapists provide 24 hour care.

Camelback Hospitals and Camelback Mental Health Center are fully accredited and licensed by the State of Arizona.

Camelback Hospitals

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Brief Summary.
Consult the package literature for prescribing information.

Indications and Usage: Ceclor® (cefaclor, Lilly) is indicated in the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

Lower respiratory infections, including pneumonia caused by *Streptococcus pneumoniae (Diplococcus pneumoniae)*, *Haemophilus influenzae*, and *S. pyogenes* (group A beta-hemolytic streptococci)

Appropriate culture and susceptibility studies should be performed to determine susceptibility of the causative organism to Ceclor.

Contraindication: Ceclor is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

Warnings: IN PENICILLIN-SENSITIVE PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE ADMINISTERED CAUTIOUSLY. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES IN WHICH PATIENTS HAVE HAD REACTIONS TO BOTH DRUG CLASSES (INCLUDING ANAPHYLAXIS AFTER PARENTERAL USE).

Antibiotics, including Ceclor, should be administered cautiously to any patient who has demonstrated some form of allergy, particularly to drugs.

Precautions: If an allergic reaction to cefaclor occurs, the drug should be discontinued, and, if necessary, the patient should be treated with appropriate agents, e.g., pressor amines, antihistamines, or corticosteroids.

Prolonged use of cefaclor may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

Ceclor should be administered with caution in the presence of markedly impaired renal function. Under such a condition, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Usage in Pregnancy—Although no teratogenic or antifertility effects were seen in reproduction studies in mice and rats receiving up to 12 times the maximum human dose or in ferrets given three times the maximum human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

Usage in Infancy—Safety of this product for use in infants less than one month of age has not been established.

Adverse Reactions: In clinical studies in 1493 patients, adverse effects considered related to cefaclor therapy were uncommon and are listed below.

Gastrointestinal symptoms occurred in about 2.5 percent of patients and included diarrhea (1 in 70) and nausea and vomiting (1 in 90).

Hypersensitivity reactions were reported in about 1.5 percent of patients and included morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs tests each occurred in less than 1 in 200 patients.

Other effects considered related to therapy included eosinophilia (1 in 50 patients) and genital pruritus or vaginitis (less than 1 in 100 patients).

Causal Relationship Uncertain—Transitory abnormalities in clinical laboratory tests results have been reported. Although they were of uncertain etiology, they are listed below to serve as alerting information for the physician.

Hepatic—Slight elevations in SGDT, SGPT, or alkaline phosphatase values (1 in 40).

Hematopoietic—Transient fluctuations in leukocyte count, predominantly lymphocytosis occurring in infants and young children (1 in 40).

Renal—Slight elevations in BUN or serum creatinine (less than 1 in 500) or abnormal urinalysis (less than 1 in 200). [070379R]

*Many authorities attribute acute infectious exacerbation of chronic bronchitis to either *S. pneumoniae* or *H. influenzae*.*

Note: Ceclor® (cefaclor) is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-allergic patients.

Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

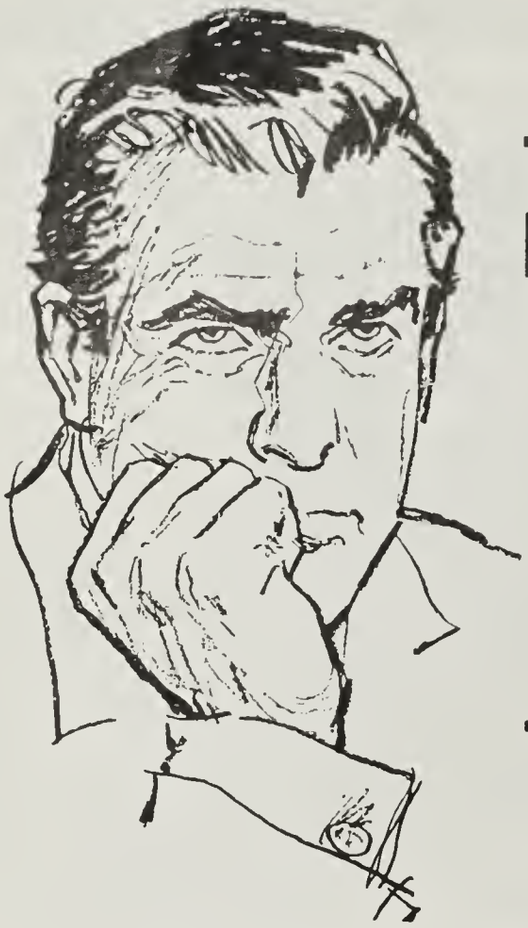
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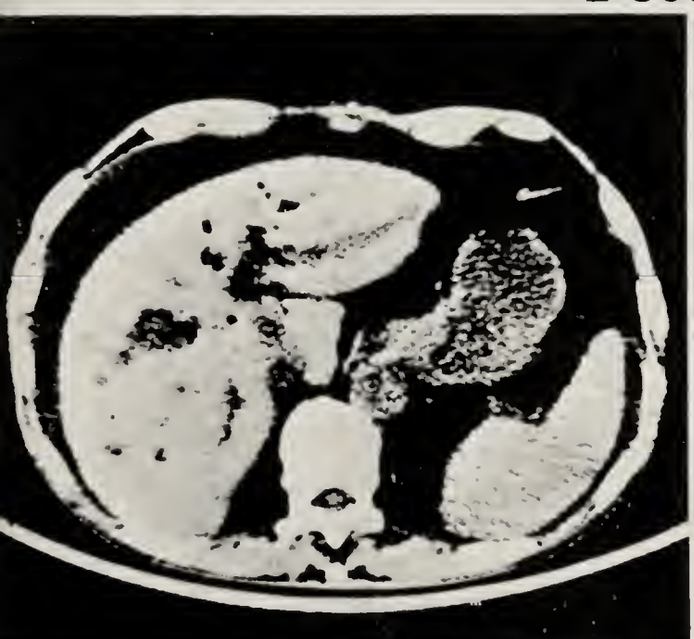
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(diethylpropion hydrochloride NF) controlled-release

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Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema, *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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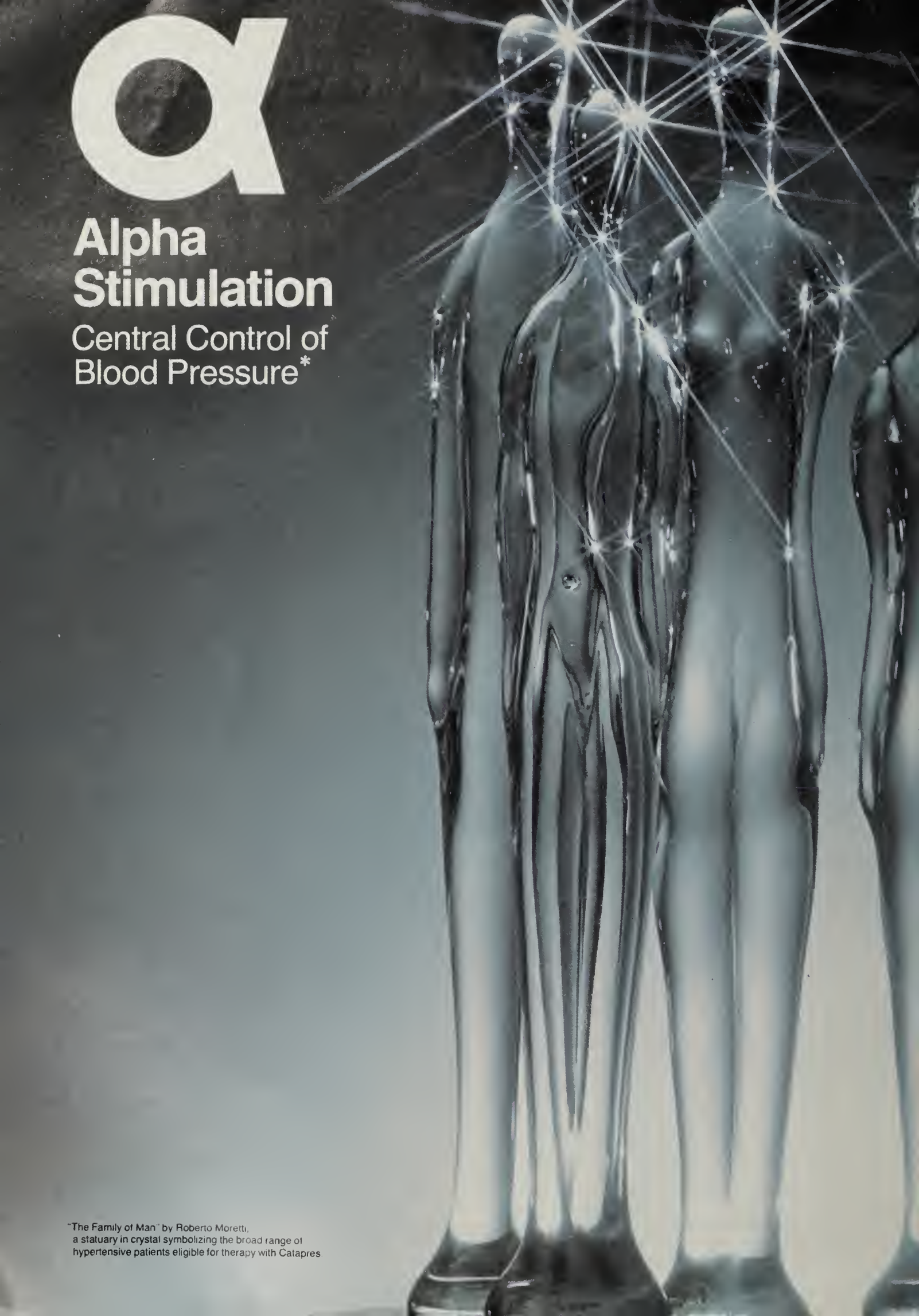
*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

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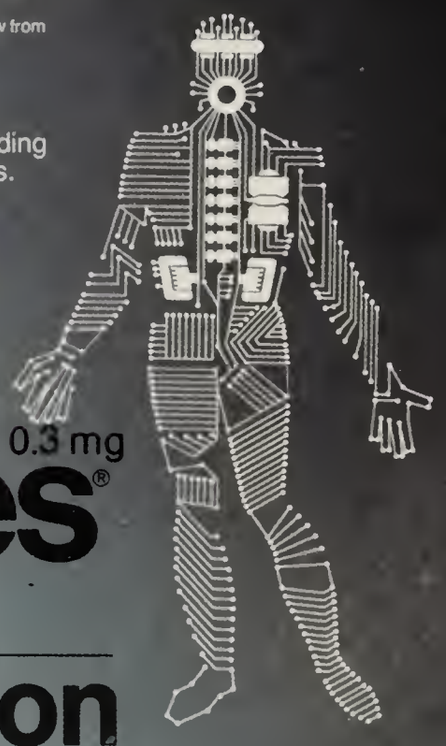
Other factors must include:

The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

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Please see last page for brief summary, including warnings, precautions, and adverse reactions.



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- Low incidence of depression, impotence, orthostatic hypotension—no fatal hepatotoxicity.
- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

For optimal results, the dose of Catapres must be adjusted according to the patient's individual blood pressure response.

Catapres®
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Tablets of 0.1, 0.2, 0.3 mg

Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In some instances an exact causal relationship has not been established.) These include. Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities in liver function tests; one report of possible drug-induced hepatitis without icterus and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlorthalidone and papaverine hydrochloride. Weight gain, transient elevation of blood glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud's phenomenon; vivid dreams or nightmares, insomnia, other behavioral changes, nervousness, restlessness, anxiety and mental depression. Also rash, angioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associated with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryness, itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomastia, weakly positive Coombs' test, asymptomatic electrocardiographic abnormalities manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or absent reflexes and vomiting followed the accidental ingestion of Catapres (clonidine hydrochloride) by several children from 19 months to 5 years of age. Gastric lavage and administration of an analeptic and vasopressor led to complete recovery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minute intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) overdosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 mg (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Also available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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Seminars in Dermatology

Melanoma, Basal and Squamous Cell Carcinomas

An Update for Clinicians

Robert Allen Schwartz, M.D., M.P.H.
Michael D. Fox, M.D.
Nagi K. Saied, M.D.

Editor:
Robert A. Schwartz, M.D.

Introduction

There have been a number of recent developments in dermatologic oncology. Those which might aid the clinician to reach the diagnosis of a disorder and recognition of its associated findings will be stressed, with therapeutic advances excluded. Dermatologic oncology for this purpose may be divided into three categories: 1) Disorders associated with obvious or suspected carcinogens (e.g. melanoma re: ultraviolet light, erythroplakia and oral squamous cell carcinoma re: chemical carcinogens in tobacco, Bowenoid papulosis re: viral and/or chemical oncogenesis); 2) Congenital or inherited diseases (e.g. nevus sebaceous, neurofibromatosis) 3) acquired conditions (e.g. epidermolysis bullosa acquisita). The oncogenic potential of human wart virus and ultraviolet light induced carcinogenesis have been the subjects of recent reviews.[1,2] In this presentation, the discussion will be limited to those types of skin cancers frequently associated with ultraviolet light induced carcinogenesis.

From: The Department of Dermatology, University of California School of Medicine, San Francisco, CA, The Section of Dermatology, University of Arizona College of Medicine, Tucson, AZ and The Department of Emergency Medicine, Chope Community Hospital, San Mateo, CA. Reprint requests to Dr. Schwartz, Dermatology Service (190), 4150 Clement Street, San Francisco, CA 94121

Melanoma

A recent study has impressively documented the fact that Southern Arizona leads the nation in the incidence of melanoma, with an incidence very close to the highest in the world.[3] In this important work, Schreiber and associates tabulate the incidence of melanoma in whites in sundrenched Southern Arizona. They repeated, with impressive vigor, the work done by Schreiber and others some ten years ago in documenting the very high incidence of nonmelanoma skin cancers in Southern Arizona.[4] By their calculations, the "crude" incidence rate of melanoma has increased from 6.49/100,000 in 1969 to 28.57 (27.20 standardized) per 100,000 in 1979, reflecting a fourfold increase in the past ten year period!

In their retrospective study, the highest incidence of melanoma was found in the age group of 50-59 years (24%), with the most common site of occurrence on the back (31%).[3] They attributed this high and increasing incidence of melanoma to meteorologic and geographic factors which provide Tucson with more annual sunlight, more clear days, and less daytime cloudiness than any populated geographic area in North America.

A recent study of caucasian Hawaiians also documents the increasing incidence of melanoma there.[5] The increased incidence of melanoma after peaks of sunspot activity may also be highly significant.[6] It should be noted that sunspot activity modulates the amount of carcinogenic ultraviolet light which passes through the stratosphere by determining the thickness of its ozone layer.

It has been pointed out that tumor thickness, as a guide to surgical management in patients with melanoma limited to skin, may be more valuable than histologic leveling.[7] Whether or not this is true, both tumor thickness microstage and Clark's level of histologic invasion should be determined on all melanoma specimens. The extremely good prognosis for thin melanomas (i.e. those less than 0.76 mm thick) may be tempered by cutaneous signs of tumor regression, the clinical hallmarks of which are focal areas of white, pink and translucent gray. This was found in a recent study to be an ominous prognostic sign and an important caveat to the histologic staging system which equates "thin melanomas" with favorable prognosis.[8] In their study, Gromet, Epstein and Blois found in a six year period that five of 23 "thin melanomas" with regression metastasized. Surprisingly, another recent study found that white, depressed areas of regression were without prognostic value.[9]

When one diagnoses a single cutaneous melanoma, one should look for more. From a study of 712 melanoma patients, 5.3% were found to have more than one primary melanoma, with 32% of these occurring simultaneously and 45% being noted within one year.[10] Based on microstage evaluation, the prognosis of these patients correlated with that of the most advanced primary lesion. Of this group of 38 patients with multiple primary melanomas, only four could be categorized as familial origin to fit the so-called "B-K

mole syndrome." This autosomal dominant syndrome is characterized by the continuous appearance of acquired moles even beyond the age of 35 years. Typically, there are from 10 to over 100 hapazardly colored lesions, frequently 5-10 mm or larger in diameter, and irregular in outline.[11] Recognition of familial melanoma is obviously highly important to both patient and family.

Another important recent melanoma study has shown that the thorax is the initial site of metastases, occurring in 12 of 13 patients in this University of California Melanoma Clinic series.[12] Since early surgical resection of metastases correlates with longer patient survival, the metastatic evaluation of melanoma patients must include a thorough thoracic examination—by either computerized tomography or conventional full lung tomography. Routine chest x-rays are not adequate! In 83% of these patients, only the thorax metastases were present; thus the thorax must be considered the early site of predilection of malignant melanoma.

Another caveat is that an "itchy" mole deserves a biopsy.[13] Epstein points out that a persistent and annoying mild pruritus is a significant clue—nearly one-half of melanoma patients are said to have encountered it.[13] Our experience here in Arizona has called attention to the fact that melanomas may appear quite verrucous, at times simulating seborrheic keratoses.[14] The so-called verrucous melanoma may merit a separate category to emphasize this presentation as well as the difficulty encountered at times in its histologic microstage evaluation.

Lip Cancer

The lower lip is a site which receives a large amount of actinic exposure. In a large series of lip cancer patients from M.D. Anderson Hospital in Texas, it was shown that squamous cell carcinoma of the lip in patients under 40 years of age has a significant metastatic potential, with at least 12 of 56 patients (21%) succumbing.[15] We too have seen a number of young patients with metastasizing lip squamous cell carcinoma. It is suggested that physicians in sunbelt regions such as Arizona be sure to examine this site. Although sun-induced squamous cell carcinoma generally has a low metastatic potential, it increases significantly in the lips. It is important to treat actinic cheilitis, to biopsy it if there is any question of progression from this cutaneous dysplasia called actinic cheilitis, and to urge that lip screens be used in addition to sunscreens. A recent publication points out that one must consider necrotizing sialometaplasia in the differential diagnosis of lip cancer, both clinically and histologically.[16] This self-healing, probably ischemic, disorder of the minor salivary glands may present as an ulcerating lesion on the lip or within the oral cavity, sometimes simulating squamous cell carcinoma.

Cutaneous Squamous Cell Carincoma

After renal transplantation, the incidence of squamous cell carcinoma (SCC) of the skin has been found to increase markedly, to 36.4 times more frequent than expected.[17] This incidence was noted to be higher in skin areas more exposed to sunlight, and to increase with time after transplantation, with multiple squamous cell carcinomas often appearing. The aggressive behavior of apparently sun-induced cutaneous SCCs in lymphoma patients has recently been emphasized.[18,19] Obviously, immunosuppressed patients must be regarded as at increased risk of this ultraviolet light-induced skin cancer.

The risk of cutaneous squamous cell carcinoma in patients treated with oral methoxsalen photochemotherapy for psoriasis also merits inclusion. A recent multicenter study shows that these patients are at increased risk of cutaneous SCCs.[20] However, there is debate about the validity of this study;[21] further work in this area should be forthcoming.

We have noted that, at least in Arizona and California, the practitioner should be aware that early papules of disseminated coccidioidomycosis may resemble actinic keratoses.[22] Since the use of ketoconazole by John Galgiani, M.D., at the University of Arizona and by Cortistaan Brass, M.D., at Stanford University has shown that it is an effective oral therapy lacking the severe side effects of amphotericin B,[23] this diagnosis, even without nervous systemic involvement, may merit therapy.

Basal Cell Epithelioma (BCE)

Bluish-gray cystic nodules on the face must be suspected of being cystic basal cell epitheliomas. In a recent discussion, it was pointed out that although the current literature emphasizes that blue-gray cystic lesions are often benign appendageal tumors, they may well be BCEs, especially if there is evidence of actinic damage.[24]

A large series of patients with the basal cell nevus syndrome, an autosomal dominant condition with multiple BCEs apparently not associated with UVL exposure, was recently reviewed.[25] A number of disasterous complications were noted, including cerebral invasion and death from an uncontrolled BCE, several severe facial mutilations in young patients, neurologic abnormalities, and difficulties with the induction of general anesthesia. Hamartomatous polyps of the stomach were noted in one patient. In addition, there are two recently reported associations of this syndrome with colonic carcinoma, a linkage which may be fortuitous.[26,27]

Basal cell epitheliomas in areas of chronic x-ray damage should alert the clinician to the possibility of deeper penetrations of the x-rays and resultant carcinogenesis in underlying structures. A patient treated with x-ray therapy for hirsutism many years ago when such therapy was acceptable and frequently given by nonphysicians, developed over 100 BCEs. Under one BCE on her breasts was the fortuitous finding of a breast carcinoma, pointing out that chronic cutaneous radiation damage, or BCEs overlying thyroid, breast or

other structures, may alert the clinician to possible underlying malignancy.[28]

Basal cell epitheliomas with sebaceous differentiation may appear clinically with a yellowish hue in addition to their more typical morphology. A sebaceous pattern, when confirmed histologically, should alert the clinician to the possibility of the so-called Torre syndrome of multiple sebaceous neoplasms, keratoacanthomas and multiple low-grade visceral malignancies.[29]

Acknowledgement

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Seminars in Endocrinology and Metabolism

Hypophosphatemia

A Clinical Review

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Hypophosphatemia is recoognized as a cause for significant morbidity, especially in the critically ill patient. Since the mid-sixties low phosphorus levels had been reported with computerized laboratory panels and profiles, but little interest was given to this deficiency and its importance in patient care. With time, it became more evident that hypophosphatemia is a basis for not only morbidity but mortality. Prevention is of vital importance, but early recognition and correction play a major role in the treatment of acute disease processes. Hypophosphatemia usually occurs in severe illness. . .eg, diabetic ketoacidosis, acute alcoholism, gram negative sepsis, hyperalimentation, carbohydrate loading, alkalosis, acidosis, and particularly, in conditions associated with phosphate-binding antacid use or prolonged phosphorus-poor intravenous therapy.

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It is estimated that an average 150 lb (70 kg) patient contains 712 gm (23,000mmoles) of phosphorus and that 80% of this amount is in bone, with 9% found in skeletal muscle. The bulk of phosphorus in skeletal muscle and viscera is inside the cell where its average concentration in cell water is 100 mmoles/liter. The majority of intracellular phosphorus will be organic and is in the form of intermediary carbohydrates, lipids, and proteins. Just as potassium is the major intracellular cation, *phosphorus is the major intracellular anion.*

Phosphorus regulates the activity of many enzymes and relates to fuel storage and critcial energy transformations. The clincial effects of hypophosphatemia are due to deficiencies of adenosine triphosphate (ATP) and 2, 3-diphosphoglycerate (2,3-DPG) in erythrocytes. ATP is vital for energy processes of most cellular structures. . .ie, the heart and 2,3-DPG is vital for proper delivery of oxygen to tissues. When serum phosphorus is below 1 mg/dl, these two vital functions may be sufficiently impaired to produce physiologic abnormalities and neuromuscular disorders.

In adults, the normal concentration of phosphorus in serum varies between 2.7 and 4.5 mg/100 ml (0.9 to 1.5 mmoles/liter). Following the ingestion of fats and carbohydrates, this level tends to decrease slightly. A decrease may also occur with respiratory alkalosis. Increases in serum levels may be found in acidosis, dehydration, and for a short period of time following exercise.

The average adult ingests about 1 gm of phosphorus daily in the diet although this figure may vary considerably. Of the amount absorbed, 90% is excreted via the urine and 10% via the feces. Regulations of phosphorus levels in the kidney is controlled by glomerular filtration and tubular absorption. Tubular absorption, in turn, is regulated by parathyroid hormone.

Hypophosphatemia in itself does not necessarily indicate phosphorus depletion, but a serious phosphorus deficiency may be present with normal concentration of inorganic serum phosphorus levels. Natural foods contain an abundance of phosphorus, and for this reason, phosphorus deficiency does not occur too often. Large quantites of phosphate-binding antacids lead to a syndrome where patients become anorectic, complain of bone pain, and weakness. These individuals will develop a hypercalciuria along with their hypophosphatemia.

A moderate hypophosphatemia[1] (i.e. between 1.0 to 2.5 mg/100 ml) is included in the conditions listed below.

Hemodialysis	Volume expansion
Hyperparathyroidism	Aldosteronism (licorice)
Starvation	Saline infusion
Glucose Administration	Hypokalemia
Fructose Administration	Hypomagnesemia
Glycerol Administration	Gram-negative bacteremia
Lactate Administration	Insulin administration
$NaHCO_3$ Infusion	Gastrin administration
Osteomalacia	Glucagon administration
Renal tubular defects	Epinephrine administration

Pregnancy
Malabsorption
Vitamin D deficiency
Acute gout
Salicylate poisoning
Corticosteroid administration
Diuretic therapy
Androgen therapy
Recovery from hypothermia

In severe diseases of the skeletal muscle such a muscular dystrophy, hypophosphatemia is less after glucose administration.

Specific kinases exist in the liver that catalyze phosphorylation of glucose (glucokinase) to glucose-5-PO_4 and inhibit the activity of glucokinase and in this manner regulate the uptake of phosphorus and glucose.[11] In direct contrast, increasing concentrations of fructose-1-PO_4 do not inhibit fructokinase. Thusly, fructose phosphorylation is not regulated. Therefore, hypophosphatemia is more pronounced after fructose than glucose.[2]

Three processes may decrease serum phosphorus by infusion of sodium lactate: 1) lactate is converted to glucose in the liver, 2) sodium lactate may be associated with volume expansion and therefore decreases fractional phosphate reabsorption by the kidney, and 3) sodium lactate induces alkalosis.

Phosphate or calcium deficiencies or both may result in osteomalacia and are associated with a moderate hypophosphatemia.

Because of poor absorption of calcium in steatorrhea, ionized calcium concentration is decreased. Parathormone production increases. As renal tubular reabsorption of phosphorus decreases, there results a moderate hypophosphatemia. Also, deficiency of Vitamin "D" will decrease absorption of calcium by the small bowel and it decreases parathormone responsiveness of bone. Over-production of parathormone follows and hypophosphatemia as a result of phosphaturia.

After the intravenous infusion of fructose, there is a rapid decline of intracellular inorganic phosphate and enzymes are activated that rapidly deplete intracellular adenine nucleotides which are converted into inosine and, in turn, into uric acid.

In gram-negative bacteremia, hypophosphatemia of a moderate degree is found.

Epinephrine may lead to hypophosphatemia because of hyperglycemia. Therefore, after large doses of corticosteroids and also in Cushings Disease, one is apt to find hypophosphatemia. The use of glucagon may lead to hypophosphatemia and phosphaturia.[3]

As Knochel points out,[1] there are seven clinical conditions of severe or profound hypophosphatemia in which the serum phosphorus concentration may fall below 1 mg/dl.:

1. Alcoholic withdrawal
2. Diabetes mellitus
3. Pharmacologic PO^1 binding
4. Recovery/diuretic phase after severe burns
5. "Hyperalimentation"
6. "Nutritional recovery syndrome"
7. Severe respiratory alkalosis

Morbidity may be caused by binding phosphorus with either magnesium hydroxide, aluminum carbonate gels, or aluminum hydroxide used in the treatment of peptic ucler disease. It also occurs in patients with chronic renal failure.

In severe third-degree burns, salt and water are retained. With healing, water and salt are mobilized and excreted carrying with them considerable phosphate.

Hyperalimentation without adequate phosphorus causes severe hypophosphatemia.

Another example of severe hypophosphatemia was seen in World War II in liberated prisoners of war when the "Nutritional Recovery Syndrome" occurred in those with severe protein-calorie malnutrition. Over-feeding with simple carbohydrates was followed by hydrothorax, ascites, edema, and death.[4]

Intense hyperventilation causes levels of 0.5 mg/100 ml. of serum phosphorus. With respiratory alkalosis, CO_2 rapidly leaves the intracellular space. The following increases of intracellular pH activates glycolysis to increase the formation of phosphorylated carbohydrate compounds within the cell.[12] The source of this phosphorus is the readily diffusible inorganic phosphate pool, and then serum phosphorus falls resulting in a fall of excretion of phosphorus into the urine.

In poorly controlled diabetes mellitus, there is a great loss of phosphate into the urine. Metabolic acidosis also may cause phosphaturia as acidosis inactivates organic phosphate inside the cell and there results a loss of inorganic phosphate into the urine.[5]

Hypophosphatemia is present in 50% of hospitalized alcoholics secondary to poor nutrition, diarrhea, vomiting and the use of antacids. However, it appears only after the appearance of hypomagnesemia. Phosphaturia is a feature of experimental Mg^{++} deficiency and leads to phosphorus deficiency.[6] Most chronic alcoholics have a low serum calcium at the time of hospital admission. . .eg, 8 to 8.5 mg/100 ml. Also in this group of patients, ketonuria is common because of inadequate diet.

There results five serious deficits with severe hypophosphatemia:

1. CNS dysfunction
2. Rhabdomyolysis
3. Platelet dysfunction
4. Red cell dysfunction
5. Leukocyte dysfunction

In hypophosphatemia, triose-phosphate values rise from a normal of 8 mmoles/ml to 300-600 mmoles/ml because inorganic phosphate is a cofactor necessary for activity of glyceraldohyde 3-phosphate dehydropgenase (G-3-PD) that facilitates conversion of triose-phosphate to 1,3,-diphosphoglycerate (1,3-DPG). Erythrocyte glycolysis is then depressed and then the formation of 2,3-DPG and adenosine triphosphate. The effect of the drop of ATP causes: 1) a decrease of red cell hexodinase activity and slows the formation of glucose-6-

phosphate, and 2) also a low ATP increases the action of phosphofructokinase with a final increase of triose-phosphates. Hypophosphatemia will cause red cells to become spheroidal, dehydrated, rigid, and entrapped in the spleen. Patients with malnutrition become glucose intolerant and develop hyperglycemia as seen in those receiving hyperalimentation.

Hypophosphatemia will cause a 50% depression of phagocytic, chemotactic, and bactericidal activity of granulocytes.[13] The method by which this impaired function takes place is by impairment of ATP synthesis.

As for CNS dysfunction,[7] symptoms such as muscular weakness, confusion, numbness, irritability, apprehension, dysarthria, obtundation, seizures, and coma may occur. The drop in red cell 2,3-DPG is important where oxygen is necessary for energy production, e.g. in the brain where oxidation of glucose via the Krebs cycle is necessary for synthesis of ATP.

Phosphate deficiency causes skeletal muscle dysfunction. In severe chronic alcoholism when the serum phosphorus is less than 1 mg/100 ml for a few days, a sharp rise in serum creatinine phosphokinase (CPK) activity occurs. CPK leaks out of the cell and there is frank cellular destruction as the muscle cells of the alcoholic are at the threshold of structural dissolution and there results rhabdomyolysis.

Lactic acidosis occurs in some diabetic patients and some alcoholics with hypophosphatemia.[8] In diabetic acidosis; hypophosphatemia reaches it nadir in from 24 to 36 hours, in chronic alcoholism; the second through the fourth day, and in those receiving hyperalimentation; on about the tenth day after beginning therapy. In the malnourished alcoholic, red cell 2,3-DPG may be depressed by phosphorus depletion and a superimposed respiratory alkalosis eliminates the Bohr effect.

In treatment, the administration of phosphate salts may cause: 1) hypophosphatemia, 2) metastatic deposition of calcium phosphate, 3) hypocalcemia, 4) hyperkalemia, 5) dehydration, and 6) hypernatremia. Therefore, contraindications of the use of phosphate salts are: 1) hypophosphatemia, (a) renal failure, (b) hypoparathyroidism; 2) hypercalcemia; 3) oliguria; and 4) tissue necrosis.

The following list of substances contain phosphorus available for treatment:

1. Milk - skim or low fat milk. Each quart contains 1000 mg of calcium and phosphorus (33 mmoles). Give slowly to the cachetic or starved patient.
2. NaH_2PO_4 - Na_2HPO_4 - give orally 15-30 cc. three times daily (it often produces diarrhea).
3. KH_2PO_4-K_2HOP_4 - available in 10 cc. vials to be added to intravenous fluids.

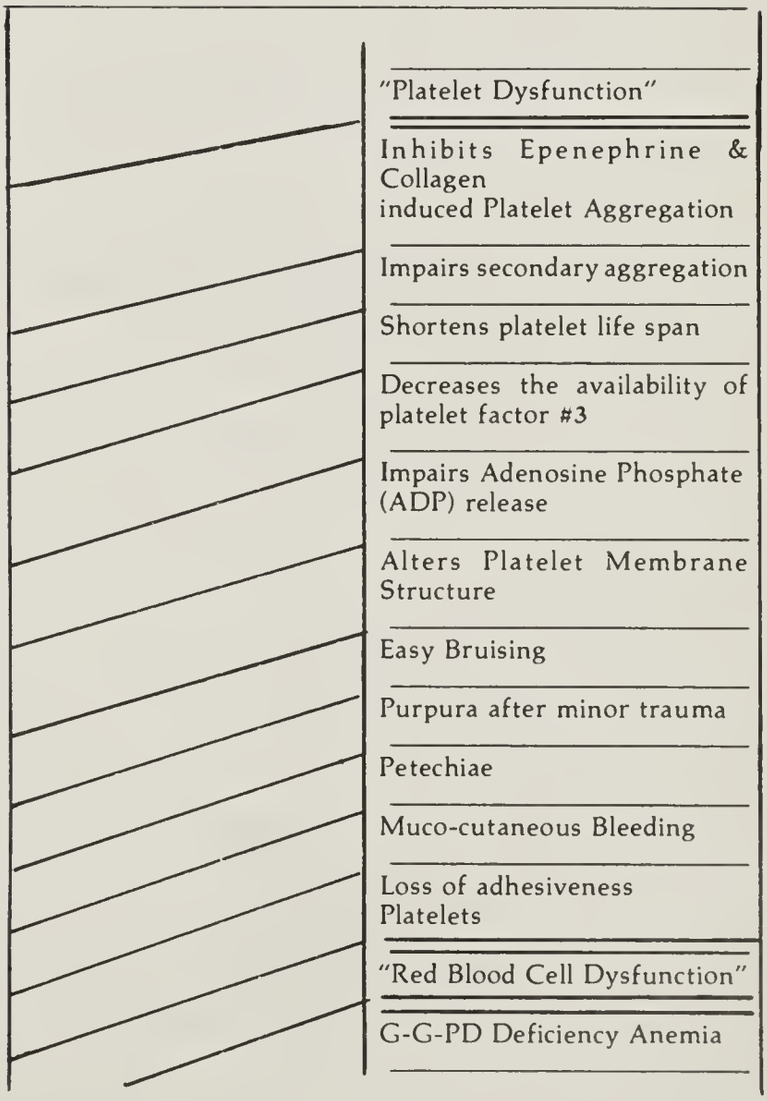
Patients with hypophosphatemia also require potassium and magnesium.

Summary

Prevention of hypophosphatemia is the key and such control can be accomplished by careful monitoring of serum phosphorus levels. Correction of low serum levels can be brought about by phosphate replacement by mouth or by vein and the correction of underlying illnesses. In this manner, deleterious effects to the skeletal system, cardiovascular system, neuromuscular system, blood system, and hepatic system will be avoided. Hypophosphatemia is a common finding in hospitalized patients. Gilbert, et al[9] in Birmingham, Alabama reported an incidence of 21.6% in a population of 18,088 inpatients. In a 100 patient study, Juan, et al[10] found that intravenous carbohydrates accounted for 45% of hypophosphatemia whereas 7% were attributable to diuretics, hyperalimentation, and alcoholism. Both insulin and hyperglucagonemia enhance the influx of phosphate from extracellular compartments into cells. It is mandatory in high risk patients that serum phosphate be monitored because low levels depress mycardial contractility, impairs bactericidal defenses, and decreases red blood cell 2,3-Diphosphoglycerate (impairment of oxygen transport to tissues).

There follows flow sheets covering signs, symptoms, laboratory and treatment which are excellent for monitoring.

Hypophosphatemia
Flow Sheet
Signs and Symptoms



Hypophosphatemia
Flow Sheet
Signs and Symptoms

Atpase Deficient Anemia

Hemolytic Anemia

Spheroidal RBC

Dehydrated RBC

RBC entrapped in spleen

Decreased red cell ATP

Depression of RBC 2,3-DPG

"Leucocyte Dysfunction"

Decreased Activity of Granulocytes

Phagocytosis 50% depression

Chemotactic resulting in Bacteriacidal serious infections

Hypophosphatemia is most pronounced in:

a. Diabetic acidosis in 24-36 hrs.

b. Chronic Alcoholism on 2nd-4th day

c. Hyperalimentation on 10th day

"Central Nervous System"

Muscular Weakness

Confusion

Numbness

Irritability

Apprehension

Dysarthria

Mental Obtundation

Apnea

Ataxia

Convulsions

Seizures

Disorientation

?? Hyperventilation

Paresthesias → mouth
\ extremities

Coma

"Rhabdomyolysis"

Myalgia

Muscle Pain

Weakness

Muscular Dystrophy

Bone Pain

Skeletal Muscle Dysfunction

Dissolution of Muscle

Hypophosphatemia
Flow Sheet
Laboratory

Date

Time

Serum Phosphorus

Serum Calcium [Ca^{++}]

Serum Chloride

Serum Magnesium [Mg^{++}]

Serum Glucose

Serum Insulin

Serum Potassium [K^{+}]

Serum Acetone

Parathyroid Hormone

Arterial Gases

Serum Sodium

Blood Chemical Profile$_{16}$

Creatinine

Protein Electrophoresis

Serum Protein

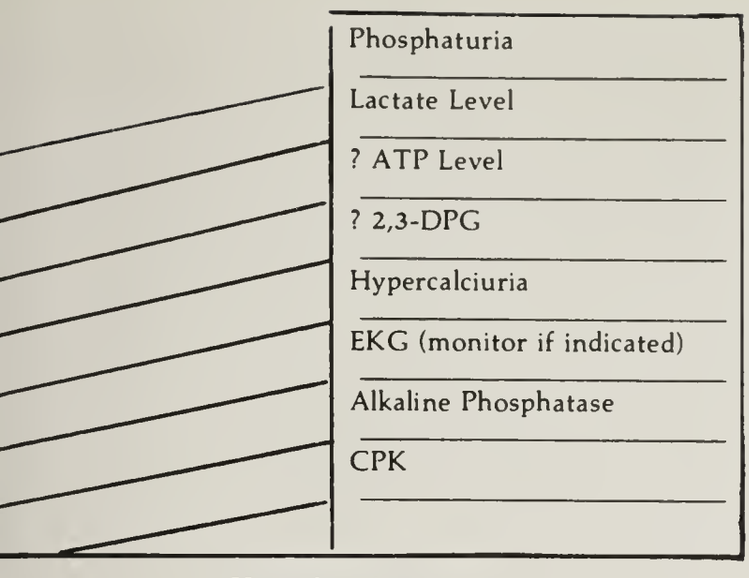
CBC

Hct. & Hb.

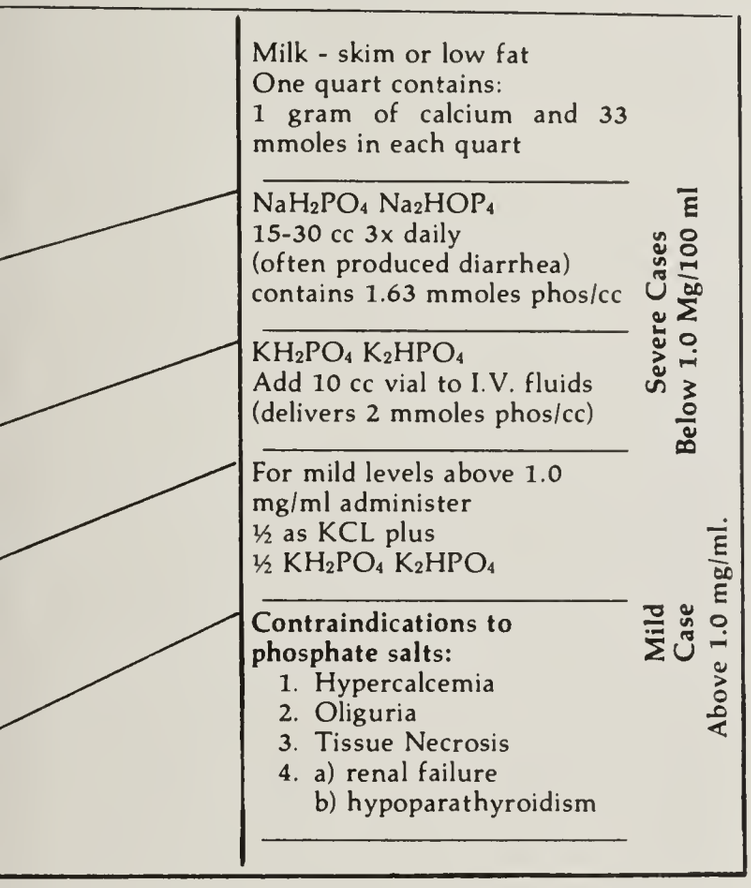
Urinalysis

Urine Ketones

CO_2



Hypophosphatemia
Flow Sheet
Treatment



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Seminars in Gastroenterology and Liver Disease

Diffuse Esophageal Spasm

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Diffuse esophageal spasm (DES) is a motor disorder of the esophagus which is an important consideration in the differential diagnosis of both dysphagia and chest pain. Although it is classically characterized by specific clinical, radiologic, and esophageal manometric findings, not all of the classical symptoms and findings are present in every patient. The following is a discussion of DES, including symptomatology, diagnosis, and treatment.

Pathophysiology and Etiology

The cause for esophageal spasm is generally unknown although some cases occur secondary to gastroesophageal reflux with esophagitis. Unlike achalasia, there is no decrease in the number of ganglion cells in Auerbach's plexus. Although there is evidence of muscular thickening in the lower 2/3 of the esophagus, there are no microscopic abnormalities noted on light microscopy. The hypersensitivity of some patients with idiopathic DES to cholinergic agents has raised the possibility that idiopathic DES may be a precursor to achalasia.

Clinical manifestations

DES is characterized symptomatically by substernal chest pain and dysphagia. While lesions causing esophageal obstruction are generally associated with dysphagia for solid foods, dysphagia with DES is characterized by difficulty with ingestion of both solids and liquids. Frequently symptoms will be induced by ingestion of hot or cold liquids. Episodes of dysphagia may not be associated with attacks of chest pain and, furthermore, chest pain may be entirely absent. The chest pain is characterized as substernal and frequently radiates to the back. The intensity of the pain is often severe and will frequently be confused with angina pectoris, especially in those patients who do not have dysphagia. Unlike anginal chest pain, there is rarely radiation to the upper extremities. Symptoms are generally intermittent in nature and do not progress with time. Weight loss is uncommon. Physical examination reveals no characteristic findings.

X-Ray

Chest x-ray is unremarkable. Esophagogram will frequently reveal isolated incoordinated contractions of the lower 2/3 of the esophagus, i.e., tertiary contractions. This gives the appearance of the so called "cork-screw" esophagus. It should be noted that barium swallow will frequently be entirely normal.

Manometry

Esophageal manometry is a study whereby pressures, and hence contractions, can be sequentially recorded within the esophagus using a triple lumen catheter which is constantly infused with water. Esophageal manometry is both more sensitive and more specific for

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esophageal spasm than barium swallow. Characteristically in DES there will be evidence of increased amplitude and duration of esophageal contractions. Furthermore, many, if not most, esophageal contractions occur simultaneously, rather than sequentially in a peristaltic fashion. In addition, the lower esophageal sphincter will occasionally manifest a higher resting pressure. However, unlike achalasia, the lower esophageal sphincter will relax normally with swallowing. Because of cholinergic hypersensitivity, subcutaneous injections of Mecholyl will frequently induce spasm on manometry as well as occasionally reproducing chest pain. However, this response is neither sensitive nor specific enough to aid in diagnosis.

Other Evaluation

Since lesions infiltrating the esophageal wall or obstructing bolus propagation can cause radiographic findings suggestive of spasm, patients suspected of having DES should undergo endoscopy. Furthermore, this allows one to exclude underlying esophagitis, which as noted previously, can be a cause for secondary esophageal spasm.

Treatment

Management of DES is virtually always medical. The patient should be instructed to concentrate on not eating rapidly. Avoidance of very hot or very cold liquids and occasionally carbonated beverages may be helpful. Patients with underlying esophagitis should, of course, have treatment directed at that problem, including antacids, antireflux maneuvers, and occasionally cimetidine. Both short and long-acting nitrates may be useful. Their effectiveness appears to be much greater in primary DES than to DES occurring secondary to esophagitis. Patients who do have symptomatic relief with sublingual nitroglycerin will generally respond to long-acting nitrites. Because of this symptomatic relief with nitroglycerin, this response cannot be used to differentiate DES from angina. Occasionally dicyclomine given before meals and at bedtime will be helpful. In those occasional patients with severe symptoms unresponsive to medical treatment described above, pneumatic dilatation will be useful. Very rarely, long myotomy of the lower esophagus will be necessary. It should be emphasized that pneumatic dilatation and particularly surgery should be reserved for only those patients with very severe symptoms who have clear documentation of primary DES and lack of response to medical therapy as described above.

Summary

Diffuse esophageal spasm is an esophageal motor disorder which has its main importance in the differential diagnosis of chest pain and dysphagia. Evaluation for these problems with respect to DES should include barium swallow, esophagogastroscopy, and, most importantly, esophageal manometry. Medical treatment is frequently though not always effective. Nevertheless, since symptoms are intermittent and non-progressive, surgery is rarely required.

Obstetrics and Gynecology

Experience with Carbon Dioxide Laser in Female Genital Neoplasia

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An almost epidemic rise in cervical intraepithelial neoplasia and condylomata acuminata has been noted in recent years. Factors influencing this could include a change in sexual mores, mass Papanicolaou screening and diagnosis of early lesions by colposcopy. With increased knowledge of cellular biology and pathologic anatomy came a marked trend toward conservatism. At present surgical "cold knife" conization of the cervix and hysterectomy are considered by some to be overtreatment for cervical intraepithelial neoplasia (CIN). An ideal form of therapy should accomplish precise and complete destruction of the lesion with no scattering of neoplastic tissue. There should be minimal damage to adjacent normal tissue and sealing of lymphatic channels to inhibit metastatic spread. Bleeding and scarring should be minimal, since quick healing and patient acceptablility are of major importance. The carbon dioxide laser meets all these criteria.

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Laser is an acronym for Light Amplificaiton by Stimulated Emission of Radiation. Schawlow and Townes working at Columbia University produced the landmark thesis on lasers in 1958.[1] The first working CO2 laser was built by C.K.N. Patel at Bell laboratories in 1964.[2] All lasers emit light. The radiation is coherent (all rays are in phase in time and space), monochromatic (same wavelength and color) and collimated (the rays are parallel). The first laser, using a ruby, produced a beam which was emitted in bursts or pulses, lasting a fraction of a millisecond. Ketcham reported the possibility of impacting viable tumor cells deeper into tissue as a result of the pulsing action, thus lessening its value in the treatment of neoplastic disorders.[3] The CO2 laser is the most efficient gas laser yet developed. The radiant energy has a wavelength of 10.6 microns, is invisible to humans and is found in the infra-red portion of the electromagnetic spectrum. The CO2 laser operates as a continuous wave device with power output as high as 500 watts or more. The rays are focused by a lens to a very small spot size. The power density can be calculated by the formula, Power Density = $\frac{\text{watts} \times 100.}{(\text{spot size in mm.})^2}$[4]

When the laser beam impacts tissue, the zone of destruction so created has a "bullet" like shape or gaussian distribution. Thus the power will be strongest in the center and fall off toward the edges. This is a very important consideration in using the laser as a surgical instrument to remove a lesion. Because the spot size is less than 2 mm it is impossible to measure the power density across the beam. It is more important to know the average power, because the laser beam moves and does not remain stationary. The energy from the laser is usually totally absorbed within a depth of 90 microns. This coupled with the fact that there is but little lateral scattering makes the new "light knife" an excellent device for exact tissue removal. While this method of treatment lends itself particularly well to cervical neoplasia there are special requirements which must be met. An expert colposcopist must identify the entire boundary and geography of the lesion, which must be verified with histopathologic examination. There must be no endocervical involvement, for if present this is a contraindication to laser therapy. The method of destruction should be well understood because treatment to a depth of 5-7 mm is usually necessary. Bellina has stated the depth of CIN involvement in crypts to be 4 mm, so treatment of 5-7 mm should ablate the lesion.[5] In the treatment of CIN the depth of destruction then should involve as much as a shallow "cold knife" conization. If these rules are followed a 95% or greater cure rate should be expected for CIN. Anderson found the depth of involvement of CIN in 99.7% to be less than 4 mm.[6] Failures might be expected if the laser treatment is too superficial or if it is an interrupted type of destruction. Upon impaction of tissue a furrow is formed and viable tumor tissue could persist between the zones of destruction. It is important to use the laser in a continuous fashion in the manner of a grid, first horizontally, and then cover the same area in a vertical fashion. To insure proper ablation, the final step should be to move the laser over the lesion in a diagonal manner. The entire transformation zone up to the squamo-columnar junction should be removed. This gives better results, for CIN is often multifocal and simple removal of isolated lesions will leave behind residual tissue that continues to have malignant potential.

Table 1

Histology	Cervix	Vagina	Total	Failures
CIN II	7		7	0
CIN III	6		6	0
CIS	3	1	4	0
Total	16	1	17	0

Using the laser generally provokes less bleeding than comparable therapy with a scalpel. There is less necrosis than with electrocautery or cryosurgery. As a result the patient has less annoying vaginal discharge. Besides sealing the small veins and lymphatics and thus reducing blood loss, the area treated is sterilized as well, reducing the problem of infection. There is virtually no postoperative pain and no visible scar formation. The lesion heals by reepithelization. Another important consideration of this therapy is that the squamo-columnar junction is neither destroyed nor displaced upward into the cervical canal, as it is commonly with surgical or cryosurgical removal. Colposcopy remains an important aid in follow-up which should be done every three months for the first year and then twice a year thereafter. If a lesion persists it will be found quickly, usually within 3-4 weeks. Our experience in laser therapy has included cases of neoplasia of the cervix and vagina as well as large condylomata acuminata and a congential vaginal septum. Twenty patients with CIN have been treated with the carbon dioxide laser. Seventeen have had complete ablation of the transformation zone, and though the follow-up period is short there has not been a single recurrence. (Table 1). The ages varied from 22 to 38, and parity did not enter into the diagnosis. Three patients with CIS were 26, 27, and 28 years of age, and all were multiparous. The depth of destruction was carried out between 5 to 7 mm.* The spot size used was 1.5 mm and the wattage from 25 to 30. Bleeding was not a problem and the only discomfort noted was a moderate vaginal discharge for the first 36 hours post treatment. Healing was noted to occur by reepithelialization within two to three weeks. The patients were instructed to refrain from coitus and douches for two weeks. Anesthesia was used in the first two patients only because of unfamiliarity with the

*The depth of laser therapy was measured by the use of a micro scale viewed through the colposcope.

technique. Since that time no anesthesia has been used except for the vulvar and vaginal lesions treated. Two patients with condylomata acuminata were treated. One had no response to podophyllin and cryosurgery, the second was treated during pregnancy. Both patients were uncomfortable for the first 24 hours and required moderate pain medication. The use of "Instant Ocean" (a commercially available saline solution) greatly helped both in pain relief and in keeping the areas clean. Within two weeks the vulvar areas were virtually healed without disturbance of normal anatomy. The 19 year old nulliparous patient with vaginal CIS (not DES exposed) was treated under anesthesia. It was necessary in order to expose the fornices to accomplish complete ablation of the lesion. The entire vaginal lesion was vaporized to a depth of 3-4 mm. Healing has been complete with no scar formation and Papanicolaou smears have remained negative. Another patient with a large congenital vertical vaginal septum was treated by excision with the laser. With the precision of the laser beam the septal tissue was removed from immediately beneath the urethra as well as from the posterior vaginal wall. Bleeding which occured posteriorly had to be separately ligated. Blood loss was estimated to be 20-30 cc. The patient had no postoperative discomfort, and healing without scar formation was complete within three weeks. The vaginal tube is now normal and pliable and there has been no dyspareunia.

Summary

The carbon dioxide laser is new and can be used effectively for therapy of many gynecologic lesions. Expertise both in the use of this instrument as well as in colposcopy is of utmost importance. The advantages of laser treatment far outweigh any adverse critique except for the inital cost of the unit. Patient acceptance has generally been guarded until a full description of the mechanism of action of the laser has been understood, after which there has been good reaction to this newer treatment. With strict guidelines for the types of lesions to be treated, the laser greatly enhances the ease of eradication of CIN and other selected gynecologic lesions. It may supersede other more popular methods of therapy currently in vogue.

Addendum

An additional 27 patients with genital tract neoplasia have been treated with comparable results since preparation of this report.

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Oncology

Coping Strategies for the Professional in Working with Oncology Patients

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Coping, especially that of the professional, has become a very popular topic recently (Caldwell, 1976; Freudenberger, 1977; Marvardi, 1979; Pines, 1978; Shubin, 1978). Interestingly, as the topic of cancer has received more attention, so too, how professionals cope in working within this field has been of interest to researchers as well. The purpose of this article is to examine our view of ourselves in working within a professional capacity with oncology patients, and to offer some guidelines which may enhance our coping abilities.

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General Psychosocial Oncology Problems

What variables contribute to the professional's difficulty in working with oncology patients? Are there special problems presented by an oncology population that differ from those with other patient populations? In view of these questions, let us examine several situations that might occur in working with an oncology population.

During an initial visit, a patient remarks, "My friends tell me to make this a learning experience—I can think of other means for learning. Would *you* choose cancer as a means for growth?" Or, in another situation, a patient says, "My doctor said that I failed on chemotherapy. . .Imagine *-me* - ! It's not me, is it? I thought it was the *drug* that failed!" And in still another situation, a patient comments, "I've tried to assume responsibility for my illness—I really have. I've been through psychotherapy, I've changed my diet, I've learned relaxation and stress management techniques, and now a recurrence—what's next?" In another situation, you receive a phone call from a family member or concerned friend. The person says to you, "Doctor, I just don't know what to do. The children are placing demands on Sally—wondering why there isn't pop or snack food in the house. Her husband is avoiding her—not arriving home until midnight and beyond each night. And Sally keeps asking me when she is going to feel better. What should I say?"

Each one of these presenting problems could serve as the basis of an entire article. Some of the themes presented in these situations which we are facing in working with an oncology population include: a theme of coping with a chronic illness; a theme of coping with a "sick role" in that a patient, independent of how he may be feeling, is reminded that he is ill because he must come to a medical setting for treatments; a theme of "responsibility" for one's illness—especially with the recent attention given to holistic approaches to cancer and other medical problems; and a theme of death and dying that is more up-front and prevalent with oncology patients than any other patient population.

In view of these themes and in an attempt to examine how we may view our role in working with oncology patients, let us examine three assessment areas: one of personal rather than strictly professional feelings; one of role expectation; and one of skill level.

Assessing Personal vs. Professional Feelings

Frequently within a care-giver role, we are giving others permission to acknowledge and express their feelings, but we are not apt to give ourselves this same permission. As a result, the behavior of many patients is labeled as "depressed" or "anxious" rather than viewed as a normal and natural response to loss. In this regard, it becomes important to assess whether our labeling of other's behavior is a diagnostic assessment or a means for emotional and intellectual distance. In reviewing the literature, several authors (Feifel 1959; McGrory, 1978) have suggested that a person entering the medical profession does so with more fear of death than the average person. Could this also be true of the oncology health professional? Do we develop a sense of intellectualism in working with patients to counterbalance our own fears? Kübler-Ross (1971) has suggested that through an understanding of the reasons for our emotions, our defensiveness, and our conflicts about death, we become better able to contribute to the well-being of the patient. Thus, understanding and relating to our own views on death may not only assist us in feeling better about the nature of our work, but also contribute to our level of effectiveness.

An example of this relationship between feelings and behavior may be seen in the care-giver who frequently feels overwhelmed or angry within the work setting and, consequently, interacts in a strained and awkward manner with the patient and family member(s). Within this context, the care-giver may also be affecting the manner or degree to which the patient and his family cope and adapt to this illness, in that they too may experience this same uneasiness and awkwardness in their own communication patterns. Thus, the care-giver is potentially placed in a position where he is inadvertently contributing to additional stimuli in which to cope—specifically, the following: underlying issues related to his own feelings of death and loss; strained communication behaviors between himself and the patient affecting his level of effectiveness in working with the patient; and strained communication patterns between the patient and other family members in which the care-giver also interacts.

Another example of how the care-giver's uneasy feelings may be transmitted into ineffective coping behavior is within a situation where a dying patient has been referred to social services because he is observed to cry frequently or display anger at the staff. This person is generally perceived as having a "problem." Who really has the problem—the person who is crying and feels angry in anticipating his loss—or the person who feels uncomfortable with these behaviors? In this regard, one social worker has expressed the comment, "If we health-care professionals have as a goal the teaching of people to be more comfortable with their feelings and freer in expressing them so that they might learn and grow to better functioning with a diagnosis of cancer, we had better be doing the same with ourselves—social workers and all team members—without cancer." (The Breast Cancer Digest, 1979).

Identifying Expectations

Another area in which clarification is needed to effectively cope is in the defining of our role as a health-care professional. When working with a patient who is facing a potentially life-threatening illness, it becomes very important to define our own expectations. For example, are we trying to assist the patient in reaching a state of "enlightenment" before death? In working with an oncology population, we are more apt to see a greater number and intensity of problems rise to the surface that have typically remained dormant for many years of

a person's life. In this regard, it becomes more of a task in assisting the patient to identify and set priorities for the different presenting problems. Perhaps the primary role of a physician or nurse is not to intervene with psychosocial issues, but in working within this field of medicine we are confronted by patients who are grasping to resolve "unfinished business" and patients who are reflective and attempting to derive meaning from their life—past as well as present. In working directly with these areas it becomes increasingly important to assess whether we are contributing to our own feelings of being overwhelmed or feeling ineffective by determining our own level of expectation in working with these patients. Do we expect ourselves to resolve the whole gamut of emotional, philosophical, religious, and social problems that the patient may be facing? Are many of these issues that are problematic to the patient even absolutely resolvable? What is realistic? Defining our level of expectation in working with oncology patients may not only enable us to feel better about the nature of our role, but also contribute to our level of effectiveness in actually accomplishing stated goals.

Examining Skill Level: What to do for the Patient

The third area of examination is one of skill level—what to do for the patient and his family in the care-giver role. Medical training emphasizes the importance of cure and quantity of life, but until recently, has focused little attention on assessing quality of life issues as well. In this regard, many care-givers feel the need to resolve rather than acknowledge the special concerns and feelings that the patient may be experiencing. Sometimes acknowledging the emotions and feelings that the patient is expressing rather than attempting to immediately resolve the problem will be most helpful, and provide an effective means for establishing a relationship with the patient. For example, saying to a patient, "It sounds as though people projecting their beliefs and attitudes onto you, rather than understanding what you're going through, is upsetting to you. . .", instead of, "I think your friends are concerned and looking out for your best interests," may be more effective in establishing a relationship with the patient. Typically, a statement such as the latter may be one that only further angers and alienates the patient.

Generally, during a time of crisis, patients are in need of support and understanding rather than intellectualization. Frequently, the patient has effective coping strategies at his disposal, but many health care professionals don't recognize the patient's own strengths and may be projecting their own values and attitudes onto the patient. This creates a dependent relationship and one in which the care-giver may be contributing to his own difficulty in coping, especially when a number of dependent-type relationships exist across a variety of patients.

Another recommended guideline and consideration involves the awareness of semantics and its relationship to the patient's understanding and response to his illness. In working with treatment protocols that present undesirable side effects, it is crucial to allow the patient as much opportunity as possible to experience success. Many times a patient is told that he failed on chemotherapy when in reality, it is the drug that failed. This creates a potential situation where a patient may experience frustration and helplessness in that his body is not responding as he desires. A change in semantics (e.g., drug failure vs patient failure) may enhance the patient's psychological tolerance of treatment, as well as his willingness to engage in various treatment protocols.

The literature encourages the oncology care-giver to address the special meaning with which patients face their illness. Specifically, this involves assessing the patients' general understanding of their cancer, their view of themselves, and their beliefs regarding the development of their cancer. An example of the importance of addressing the patient's beliefs relates to the popular belief that cancer is contagious. This view may be transmitted into a behavior pattern where blame and guilt are placed on others, and alienation and isolation can occur at a time when social interaction and support is very important. Another popular misconception of many patients concerning their cancer is that it is immediately identifiable with death. This attitude may become transmitted into a behavior pattern where the patient stops buying clothing and household items. In this instance, the patient may also develop a speech pattern that becomes more and more death oriented. Generally, this type of patient may not respond well to medical treatment, and a self-fulfilling prophecy may result. In eliciting information related to the patient's belief about his cancer, the care-giver may reshape misconceptions or introduce new ideas which may prevent the onset of more disturbed or pathologic behaviors at a later time. In addition, attention to these factors may assist the patient and family to adapt more favorably to the changes imposed by the illness.

A final area that relates to skill level in providing quality patient care involves knowledge and awareness of hospital and community resources. You may find your ability to cope that much stronger if your are aware of the resources that are available to you and your patients and the manner in which they function. A social worker, psychologist, or psychiatrist with special expertise in communication may assist you in eliciting the questions, fears, concerns, attitudes, and beliefs of your patients. This information may be instrumental in decreasing patient fears, and may contribute to a patient's willingness and/or overall response to medical treatment. The clergy may be instrumental in meeting the religious and spiritual needs of the patient. Self-help and volunteer groups may assist the patient in feeling a sense of control and an increase in self-esteem in that these groups may show the patient that he has something to give to others. Utilizing the hospital and community resources may assist you in minimizing your

own degree of stress as a health care provider and may assist you as well in better meeting the total needs of your patients. Several authors in the literature ask if we have moved from a position of denial and avoidance to one of fanaticism in caring for the psychosocial needs of people with cancer and propose that this is an "equally potential hazard" (McGrory, 1978). Suggestions and guidelines in utilizing our technical skills emphasize that people's needs and coping patterns are uniquely individual in dying just as in living. To care adequately for a person who is facing a life-threatening illness, it is important to assess the individual and specific areas of needs, strengths, and meanings with which people face their illness (Cassileth, 1979). In recognizing and respecting the variety of ways in which people grieve, cope, and adapt, perhaps we can decrease our own anxiety about the "best method for expressing grief," and sharpen our skills in actually relating to rather than judging or strictly responding to the person who has cancer. Perhaps in this way, we will not only be providing a better service to the patient, but a better service to ourselves as well.

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Psychiatric Disorders

Emotional Problems of Physicians

III A Longitudinal Approach to the Problems

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Abstract

Educational approaches to the problems of the emotionally sick physician need to be vastly improved in medical schools, in house officer training, and in continuing medical education. One key problem is the physician's attitude toward his own emotional illness; another is the attitude of the profession toward the sick physician. Attitudinal sets of caring, concern, readiness to recognize problems, and referring emotionally disturbed students and colleagues need to be established early in the professional's life time and sustained and cultivated throughout his career. Medical School Selection Committees may need to pay more heed to vulnerable populations and select out grossly deviant candidates.

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Introduction

The key lesson in the study of emotional problems of physicians is that physicians are human beings, and as such are subject to a variety of medical and psychiatric illnesses in the course of their professional life time because of environmental and internal pressures, and critical life incidents which make some persons more vulnerable. The approaches to the problems must be broad, flexible, and ultimately specific to the needs of the individual. The ideal is prevention. Steps need to be taken toward early identification of problems, and methods developed which will enable the physician at risk to learn how to avoid "crises" or critical episodes, and how to solve problems engendered after they arise. Also, rapid appropriate treatment by a colleague needs to be instituted once clinical problems are identified.

Improved measures need to be taken along the entire spectrum of the physician's life. Improvements should begin with Medical School Admission Committees. Better education is needed for the medical student in medical school, the house officer in graduate education, and the practicing physician as part of his continuing education. Formal learning about emotional problems of physicians is important as is acquiring skills in dealing with them. The development of appropriate attitudes by the physician toward his own emotional problems and those of his colleagues is essential.

Medical School Selection

Vaillant's studies have shown that the critical variable in whether physicians have relatively poor marriages, use drugs or alcohol heavily, or obtain psychotherapy was their less stable childhood and adolescent adjustments compared to healthy colleagues.[1] It was not the choice of a medical career per se that is the critical factor in making physicians vulnerable to emotional problems. Modlin and Montes' study of physicians addicts identifies pre-addiction factors which again point to early life adjustment problems which lead to increased vulnerability to addiction.[2]

Though Medical School Selection Committees give special weight to Medical Aptitude Tests and grade point averages, insufficient attention is drawn to early life adjustments, previous physical and emotional problems, and vulnerability factors to future psychiatric problems. An argument could be made that each Admissions Committee should decide whether applicants identified with previous histories of antisocial behavior, one or more suicide attempts, drug addiction, schizophrenia, or other psychiatric problems should have equal opportunity to enter medical school as those applicants without identified problems. Ideally, this could be obtained from automated history forms. So far as possible such histories should be discussed in the same objective manner as applicants with juvenile diabetes, renal failure, rheumatic heart disease, systemic lupus erythematosis, chronic leukemia, or rheumatoid arthritis. Ideally, a comprehensive three generation health history should be obtained on all applicants with proper measures taken to protect the confidentiality of the data. Under present laws, such information could only be obtained from interviews, a highly unsatisfactory method. When vulnerabilities are identified, interviewers not responsible for the care of the applicant's illness, but knowledgeable about the identified problem should interview and, where indicated, examine the applicant. In the instances of arthritis, a rheumatologist should prepare a report, and with suicidal behavior a psychiatrist.

Since many psychiatric problems may be concealed or go unidentified, skilled psychiatric interviewers should be employed as part of the admissions process. Any criteria regarding health problems that the Admissions Committee has established should be known to the interviewer. As a minimum, gross behavioral deviation should be identified and reported. Candidates who decompensate under stress should be excluded. Ideally, histories of childhood illnesses, the quality of father-child relationships, mother-child relationship, sibling- and peer-child relationships, recreational and avocational pursuits, family histories of mental illness with special emphasis on mood disorders, alcoholism and drug dependence, as well as current marital and family relationships should be obtained.

The intelligent interpretation of this data requires keen psychological insight by the entire Admissions Committee which may be enhanced by the expertise of an experienced psychiatrist. The presence of risk factors does not automatically disqualify an applicant, particularly those showing other major strengths. Ultimately, followup by controlled studies on high risk candidates both accepted and rejected need to be conducted.[3]

Another approach would be for Admissions Committees to accept candidates who have demonstrated their ability and emotional stability to live and work with sick people. Perhaps 10-20% of each class should include former nurses, medical corpsmen, physician assistants, or other health professionals.

Medical School Experience

In Pitts' random sample of forty medical students, 7.5% were diagnosed an manic depressive, 2.5% with anxiety reaction, 2.5% with chronic alcoholism, and 2.5% as psychiatrically ill but not diagnosed.[4] Eighty-three percent of those with identified psychiatric illness, had positive family histories for psychiatric illness whereas only 25% of those without psychiatric illness had positive family histories. The most common illness in blood relatives was manic depressive illness. Of the ten students who had suicidal thoughts, three had given suicide serious consideration.

Early detection in medical school of psychiatric problems, and particularly mood disorders; is important, clearly those with clinical disorders should be referred by highly sympathetic faculty persons in a skillful, confidential manner for thorough evaluation and treatment. Medical school is the place where medical knowledge is acquired and where professional life time study habits are learned. Medical faculties are responsible for making medical students cognizant of our human foibles and vulnerabilities. Hence, as part of the study of psychopathology, when affective disorders are discussed, the psychiatric faculty member should include not only the high vulnerability to depression and suicide of medical professionals, but also that the problem is a self-limiting one, and one responsive to modern treatment modalities.

The pharmacology professors need to stress during didactic sessions on drug addiction that the medical profession is practicularly vulnerable to alcohol, narcotic and other drug addictions.[5] Also in talking about pharmacotherapeutics, an even more key lesson is that physicians should not prescribe for themselves, nor for their families. Prescriptions for physician colleagues and their families should be restricted to those instances when a doctor-patient contract has been established.

In the human behavior courses, the subject of marriage and family life should include material on the history of poor marriages of physicians and the physician's neglect of his spouse and children.[1,6] Such a forum can be used to help the medical student understand that he has two important responsibilities in life: his professional life and his personal, family life. Little emphasis needs to be given to the former because it is hammered home, implicitly and explicitly, on a daily basis. The latter should be stressed with equal importance. The subject of human sexuality and the desensitization to sexual problems should be taught in the context of the large problem.

The introduction to the topic of medicine as a high risk profession can be taught as part of human behavior courses, part of clinical psychiatry, but probably most meaningfully as part of the introduction to clinical medicine (the medical interviewing and physical diagnosis courses). It is in the discussion that follows patient-student interactions, that the clinical faculty member has an opportunity to review not only the history and physical findings, but also how the student felt about the patient. Faculty tutors must feel comfortable talking about the interpersonal aspects of the doctor-patient relationship. Unprepared faculty members will not be able to carry out this function. If such neutral questions about how the student feels can be asked with equal weight along with history and physical findings, then the student can begin to discuss the variety of reactions he may have to a variety of patients. Ultimately, the message will register that it is all right for him not to like every patient and that he is human too. Since physicians in distress are most stressed by demanding patients, insights acquired during medical school could spare the future physician unnecessary grief.

Discussion of the doctor-patient relationship, though key in the life of the physician, is not the purpose of this paper. Instead, it is written to highlight the attitudes of physicians toward their own health and that of their colleagues. Unfortunately, this is again a woefully neglected area. It is the collective responsibiility of medical school faculties and administrations to be interested, concerned, and actively involved in the physical and emotional health of medical students. Since attitudes are primarily learned through identification with respected teachers, these same attitudes about physical and emotional health can be learned by the student in regard to his own health and that of his collegues. They can be sustained beyond medical school into his professional career.

As part of the introduction to each course in medical school educational objectives, course methods, specific tasks and expectations are usually spelled out. In addition, a teacher in each department gifted in dealing with student health problems should be encouraged to talk to the class. He should stress not only his interest in students' health but also if he or any of his colleagues suspects any medical or psychiatric problems that he will feel free to discuss his observations with the student. Appropriate referrals of the student to a physician will be initiated, if he does not have one. Referrals must be conducted in a professional, competent, and confidential manner. Ultimately, students will identify the faculty members who are dedicated, gifted, and trustworthy in carrying out this responsibility. Other introductory statements can include the message that if a student is ill, the expectation is that he will not report for work, but will take appropriate steps to take care of himself. If he has clinical responsibilities, than his professional responsibility is to have his peers take calls for him. Punctuated statements like "you're no damned good coming to work sick" may get the message across most effectively.

The followup once problems are recognized is crucial. Many faculty members may not be accustomed to attuning themselves to student health or may hide behind denial, rationalization, or other defenses to not get involved. The more uniform the commitment of a faculty to this crucical area, the more effective it will be. Obviously, the other problem is practicing what one preaches in terms of faculty members caring for themselves and caring about their colleagues. The latter problem may take at least one generation of faculty members to correct.

House Officers

Many of the same principles mentioned for approaching medical students applies to house officers. Principal differences between the two groups point to the need for additional measures. Responsibility for the house officer's education switches from a medical school base most often to a hospital* base. The field of study generally narrows from the multiple medical discipline to a single one. The brief relationships with multiple faculty members in various departments is replaced by a more sustained relationship with fewer teachers.

In graduate training the house officer has many more clinical responsibilities. He gets paid a living wage for often an excessive work load. He has fewer peers in his own training program with whom he can relate than he did in medical school. The promise to spend more time with spouse and family often goes unfulfilled in the name of further education. The single physician is often most vulnerable by not having more time to take care of personal needs which he or she may have promised himself or herself while sacrificing this aspect of life in medical school.[7]

The responsibilities and attitudes of concern for the physical and emotional welfare of house officers shifts from the collective responsibility of medical school faculties to program directors of residencies, chairmen of departments sponsoring house officer training, hospital educational directors, medical teaching staffs of hospitals, and hospital administrators. With fewer numbers of teachers in any one program than in medical school, opportunity for neglect in the area of physical and emotional health is greater.

The attitudinal set described for medical school faculties applies equally to hospital teaching staffs in order to effectively deal with the emotional and physical problems of house officers. Periodic evaluation sessions for individual house officers to discuss professional growth with program directors can equally be used to discuss personal growth and identify any medical or psychiatric problems. Should the problems appear to have a direct relationship to any excessive work load or difficulties adjusting to a particular setting, then diminishing the work or changing the setting may prove to be ameliorative. If the problem is a clinical one, then appropriate referral is in order.

The stability of marriages and the emotional health of house officer's spouses and families can be addressed in spouse's groups. These same groups can serve to identify physical and emotional problems of house officers and work can be started in advising the spouse how to get help for a troubled house officer. If marital or family problems are identified, again appropriate referrals should be made.

In terms of the doctor-patient relationship, house officers should be encouraged to discuss their difficult management cases. One effective method is to have regularly scheduled group meetings of house officers to collectively discuss difficult patients with a senior clinician (in many cases a consultant psychiatrist) and tease out the component parts of what makes these patients difficult. This is recommended in addition to traditional ward rounds. This method was instituted by Michael Balint[8] and supported by Henry Brosin.[8]

Again, if bold messages are needed, warnings such as "the life you save may be your own," or threats like "you may lose your license, or be subject to malpractice suits if you don't attend to the doctor-patient relationship" can be effective in capturing the house officer's attention.

The Practicing Physician

The largest proportion of a physician's career occurs after completion of medical school and graduate training. Also, the vulnerability to emotional problems, albeit, depression, suicide, drug addiction, and alcoholism begins after age 40, years after completion of training.[2,8] Hence, it would seem reasonable to devote the greatest energies to this portion of a physicians's life. There are two reasons why this is not done.

First, the principles and approaches to the first two stages apply to this third stage. Secondly, since the principal lesson deals with attitudes, it is very difficult to change these after forty years. If one believes in his own immortality, his infallibility, or in his omnipotence for at least twenty of his last forty years, then it becomes difficult, if not impossible to change these when they become ingrained.

Hopefully, however, groups of practicing physicians will continue (if they started out doing so as house officers) to sit together on a regular basis to discuss difficult patients so that the practice of medicine does not become boring, repetitive, unexciting, lacking in rewards, no longer fun, anxiety producing, or depressing. Those deprived of previous experiences should be encouraged to participate.

Hospital staffs should form committees of experts to deal anonymously with complaints about doctors suspected of mental disorders. These expert committees should aim ultimately toward early recognition and referral of troubled physicians for appropriate treatment. They should have no disciplinary powers. The hospital administration is responsible for seeing that physicians incompetent to practice be suspended from duties they can no longer perform. However, the greater responsibility is to see that the individual physician gets help.

Group practices have similar responsibilities in

Clinic settings are implied equally when applicable in this section.

ambulatory care settings. For physicians in solo practice and those without hospital practice, local medical societies have a responsibility for such colleagues who may be suffering.

Spouse groups at various levels should be encouraged, whether in a small group practice, a hospital setting, or local or state medical societies to discuss vulnerabilities of physicians to emotional disorders. They can be organized as Brosin suggests with the title "Preserve Spouses' Club."[7]

Again, the ultimate aim is for early detection of problems and appropriate measures to correct them.

One of the most difficult tasks will be to change physicians' tendencies to not recognize, to minimize, or, most likely, to deny emotional problems of colleagues. This tradition may require a generation to change. Hopefully, new attitudes instilled in medical school and fostered in house officer training will be cultivated through the physician's continued education. Again, the legal threat of coercive measures which could limit or suspend physician's practices may have to be invoked in order to convince physicians of the importance of being active in the process of securing help for their sick colleagues.

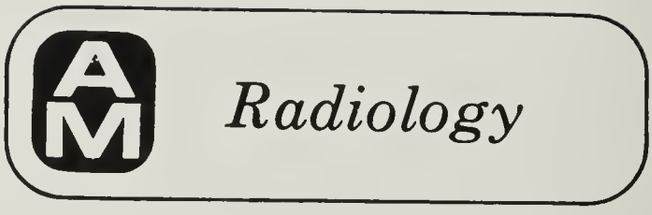
Finally, each physician must carry his share of the responsibility of recognizing a physician colleague in distress, of responding positively to distress signals from spouses or family members of a physician colleague, and ultimately of recognizing our own vulnerabilities and seeking help from fellow professionals when we or our families are in need.

Acknowledgements

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Case of the Month No. 44

Thomas Abram, M.D.
Gerald D. Pond, M.D.

A 73 year old male with a known pelvic neoplasm and secondary thrombophlebitis had a CT scan of the pelvis to detect a possible mass obstruction of the vein by the tumor.

What abnormality do you see?

From: University of Arizona Health Sciences Center, Department of Radiology, Tucson, AZ 85724.

Hematuria

Georgetta C. Bidwell, M.D.

Editors:
Georgette C. Bidwell, M.D.
George Nickas, M.D.

Hematuria is a common finding that prompts the physician to embark on a search for etiology. When hematuria is gross or painful the urgency of the situation forces one to action. When hematuria is microscopic or painless it may be missed by patient and physician alike. The goal of this paper is to focus primarily on hematuria which has a renal etiology and may signify intrinsic renal or vascular disease rather than to emphasize hematuria of lower urinary etiology.

True hematuria is blood in the urine. Red urine must be distinguished from hematuria. Certain Phenolphthalein and beet dyes may be the etiology of red urine. Also, the Dipstick UA may show positive occult blood but if the urinalysis is negative for red blood cells one must suspect pigmenturia of the hemoglobin or myoglobin type.

How much hematuria is significant? Certainly three to five red cells per high power field needs to be investigated. During fever or acute inflammatory states hematuria may occur without underlying disease. Factitious hematuria may occur in patients with psychiatric disorders and if this is suspect an observed voided urinalysis and/or catheter analysis is necessary.

Females, during their menstrual cycle, need to have a urinalysis repeated following the menses to determine if hematuria persists. Hematuria surrounding the menses may indicate endometriosis in the urinary system. This is a rare and uncommon etiology of hematuria.

If the above are excluded, three to five red cells per high power field needs an explanation. The old adage that hematuria means trauma, tumor, TB (infection) or stone still holds. In these catagories the etiology is often obvious by history or by physical examination. Infection or cystitis in young females and prostatitis in young males is symptomatic. Tuberculosis may have less symptoms early on but sterile pyuria with hematuria plus a history of TB in the past leads one to pursue the patient further with a workup to include an intravenous pyelogram and special urine cultures.

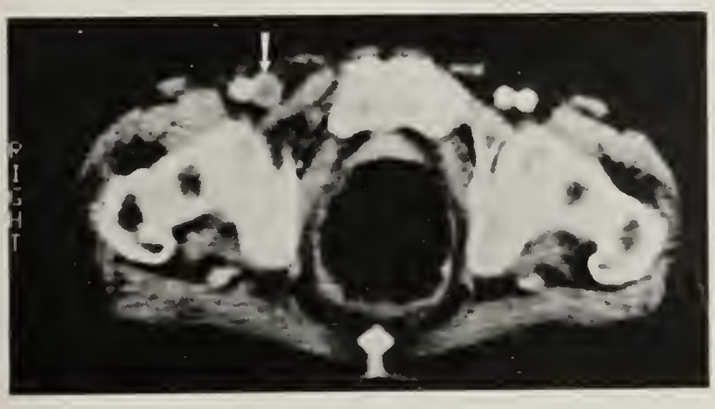


Figure 1

Continued on page 575→

From: 500 E. Thomas Road, Phoenix, AZ 85012

Tumors anywhere in the GU system may yield hematuria. Usually in an older patient the source is bladder or prostate. The ureters or kidney may also be involved. Renal cell carcinoma - the most common upper urinary tumor - may be manifested by fever, flank mass, hematuria and either polycythemia or more commonly anemia. Hypercalcemia and hyperuricemia may be associated. Renal cell carcinoma is often found first from its metastatic presentation rather than the primary site.

Stones are a very common etiology of hematuria especially in this area. It is an entity which will be discussed in a future paper.

Trauma is usually obvious from the history. Hematuria, however, may be silent following trauma until the patient presents with hypertension new in onset and hematuria several weeks later. Here, traumatic arteriovenous connections may be the etiology or an underlying induced renovascular lesion.

Now we come to the renal etiologies of hematuria that may be more insidious. A clue to renal etiology of hematuria is the urinalysis showing red cell casts, a concentrating defect or proteinuria. Protein in the urine should be quantitated and electrophoresed to determine selectivity.

Congenital or hereditary diseases cannot be forgotton as an etiology of hematuria. They include, polycystic kidneys, hereditary nephritis or Alport's syndrome and medullary sponge kidney.

In polycystic kidneys the patient may range in age from birth to the eighth decade. Hypertension with hyperuricemia is an associated finding. Other vascular anomalies may also be associated such as aneurysmal disease like berry aneurysms. Stones may also be associated. Family history is strong but not always detectable as there is high spontaneous mutation rate.

Hereditary nephritis or Alport's syndrome is a constellation of findings usually in young males. These findings include hematuria, proteinuria or renal failure, sensory neural hearing losses and lens abnormalities. Females may present a hematuria alone and are carriers of this disorder.

Medullary sponge kidney is a familial disease manifested with hematuria, recurrent stone formation and urinary tract infections. Hematuria may be macroscopic and anemia may be a common finding. This disorder is more significant for its morbidity than it is for its mortality with respect to renal functional impairment.

Vascular disease is another etiology of primary hematuria. It can be from the microvasculature such as in vasculitis, arteriovenous or an AV malformation site.

Vasculitis may be primary as in the polyarteritities or collagen vascular in etiology. The clue here is that there is usually evidence of multisystem involvement of which kidney is just one element.

An acute arterial occlusion is usually associated with pain, hypertension and significant elevation of the LDH. Arteriography is the test of choice although, scans may be helpful.

A venous occlusion—resulting from renal vein thrombosis—presents with pain, hematuria and heavy proteinuria. This may be associated with a renal lesion of membraneous nephropathy. Again, arteriography and/or venography aid in the diagnosis.

Arteriovenous malformations are difficult to elucidate and may defy definition even at the level of the arteriogram. Large ones may be picked up on the ultrasound examination.

An intersting area of renal hematuria is the entity of Berger's disease or IgA nephropathy. This disease is more common in males, has episodes of microscopic and macroscopic hematuria usually associated with or exacerbated by upper respiratory infection. It is thought to be mostly benign but occassionally renal failure occurs. The diagnosis made on biopsy with a positive immunofluorescent IgA mesangial staining. This entity is to be distinguished from benign recurrent familial hematuria where the hematuria persists without obvious evidence of any underlying disease even when renal biopsy and arteriography are performed.

Another interesting entity these days is jogger's hematuria. Although, debate still continues, most feel that jogger's hematuria rarely indicates underlying disease and usually cessation of jogging diminishes and causes the hematuria to cease. Even changing the jogging surface to a softer pavement may improve this particular problem or completely eliminate it. Usually there is no renal problem acute or chronic if good hydration is maintained following jogging.

Any intrinsic glomerular or tubular lesion can yield hematuria. Here, biopsy can make pathologic diagnosis. The clue, again, may be found in studies such as the urinalysis, creatinine or other renal function testing and occasionally radiographic studies such as the intravenous pyelogram, renogram scan or ultrasound examination.

Drugs can also produce hematuria. We are aware of anticoagulants and a fact that still holds, if isolated genitourinary bleeding occurs with anticoagulant treatment a renal lesion or lesion in the genitourinary tract must be suspected.

Other drugs such as aspirin with acetosalicylic acids (ASA) may produce GU bleeding from arteriovenous malformations not otherwise manifested. Analgesics, in general, particularly phenacetin can produce hematuria. This usually is associated with sterile pyuria and a tubular concentrating defect. Analgesic nephropathy is a topic which will be discussed in a further paper.

Finally, hemoglobinopathies such as sickle cell disease, sickle cell trait and hemoglobin SC disease may yield hematuria.

In general, an approach to hematuria includes careful history and physical examination, urinalysis with specific attention to the presence of casts, the concentrating ability and presence of protein. From there, the workup is either toward intrinsic renal disease

or vascular disease with evaluation to include radiographic studies, renogram scans, ultrasonography and even renal biopsy—especially if primary renal pathology is suspect, or focus on lower urinary pathology with the workup to include cystoscopy first. If renal primary disease is suspect then laboratory testing which includes collagen vascular screening or hemoglobin electrophoresis are in order.

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Radiology Continued

Answer: Right Femoral Vein Occlusion (Thrombophlebitis)

A constrast enhanced CT scan demonstrates failure of opacification of the vein, which can be compared to the opacified artery just lateral to it. The vein is also dilated, and this can be confirmed by comparing it to the contralateral vein, which is smaller in diameter as well as contrast-filled. These findings are the hallmarks of venous thrombosis as seen by CT.[1,2] Tumor thrombosis of the renal vein and inferior vena cava have also been identified in this fashion.[1]

Intravenous injection of x-ray contrast into a pedal vein during CT scanning has also been effective in demonstrating thrombosis of large lower extremity veins.

Faster scanning times have markedly improved resolution of these vessels. CT scanning will not likely challenge conventional venography as the primary radiographic test for diagnosis of venous thrombosis, but in selected cases it could prove to be a valuable adjunct in diagnosing or following certain venous disorders.

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Special Article

Placement of Patients in Long Term Care Facilities in Arizona

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Director, Arizona Department of Health Services

When evaluating the total complex of human services, those aspects relating to long term care of the elderly and the functionally disabled population are becoming increasingly important. The reasons are many and well documented. Advances in medical technology and improved living conditions have shifted the major burden of illness from acute conditions to chronic and degenerative diseases. The elderly are the fastest growing segment of our population and are the group at highest risk for chronic illness requiring care in long term facilities. In addition to the elderly, an increasing number of those in younger age groups have developmental disabilities or mental disorders and may, therefore, require long term care.

These trends have resulted in health policy and health care organization changes. Currently, the United States has more beds in long term care facilities than in general hospitals. Whenever possible maintenance and custodial care are being displaced by active treatment and rehabilitation. Patients placed in the long term care facilities may require services ranging from minimal oversight care for sub-acute conditions following hospitalization to total maintenance. It is essential that persons requiring long term care be appropriately placed in licensed facilities which can provide the level of care and services appropriate to individual needs.

The problem of inappropriate placement of patients in long term care facilities has been brought to the attention of the Arizona Department of Health Services. Results of a study conducted by the Center for the Law in the Public Interest and a Maricopa County Department of Health Services survey of all boarding homes under permit in Maricopa County indicated that approximately two-thirds of these facilities housed residents in apparent need of health-related or nursing care services. A special project to survey the boarding homes in Maricopa County was undertaken by the Arizona Department of Health Services in collaboration with the Arizona Department of Economic Security and the Maricopa County Department of Health Services.

On-site surveys were conducted by a multidisciplinary team composed of representatives of the three agencies. Surveys of 34 boarding homes were completed by June, 1980; team findings offered evidence in support of the original observations relating to the number of boarding home residents in need of health-related services. The total census was 459 residents in the 34 homes. Assessments of 304 residents were conducted by a public health nurse from the Maricopa County Department of Health Services and a social worker from the Arizona Department of Economic Security. They found that 180 residents, or 59%, were inappropriately placed in boarding homes, and that 82% of the inappropriately placed residents required services which should be provided in licensed supervisory care homes. The remaining 33 residents required care in licensed personal, intermediate, or skilled nursing facilities. It was also found that residents had been inappropriately placed in boarding homes by hospital social workers, hospital discharge planners, and by physician referral.

The purpose of this paper is to outline the classification for licensed long term care facilities in Arizona and to describe the various levels of care in an effort to achieve appropriate placement of residents in licensed facilities that are staffed and equipped to meet their needs.

Levels of Care

The Arizona Department of Health Services licenses three (3) categories of nursing care institutions and two (2) categories of residential care institutions:

Nursing Care Institutions: A.R.S. § 36-401.17 defines "nursing care institution" as *"a health care institution providing inpatient beds or resident beds and nursing services to persons who need nursing services on a continuing basis but who do not require hospital care or direct daily care from a physician."* Licensed nursing care institutions include skilled nursing facilities, intermediate care facilities and personal care facilities.

Residential Care Institutions: A.R.S. § 36-401.12 defines "health related services" as *"services, other than medical pertaining to general supervision, protective and personal care services or supervisory care services."* Licensed residential care institutions include supervisory care homes and recovery homes.

Boarding Homes

There has been some confusion concerning the status of boarding homes in Arizona. These homes are not licensed by the Arizona Department of Health Services. In Maricopa County, they are inspected by county sanitarians and are issued a permit when found to be in compliance with County Health Code for residences. In the other thirteen counties, there is no inspection or oversight of these homes. Boarding homes are classified in the same category as motels and hotels and are defined as *"establishments which provide domiciliary and ancillary commercial services but do not provide adaptive, medical, hospital or health-related services"* (A.R.S. § 36-402.2).

Individuals referred to boarding homes for placement should be totally independent in self-care including bathing, dressing, and eating. They should be continent of bowel and bladder or able to care for an indwelling catheter or colostomy without assistance. Such residents must also be able to ambulate without assistance, even when using a mechanical device such as a walker, or be independent in a wheelchair. In addition, medications and treatments must be self-administered without assistance or may be administered by home health agency personnel or at outpatient treatment facilities. Boarding home operators and staff may not provide health-related or personal care services; such services may only be provided by licensed facilities.

Supervisory Care Homes

A supervisory care home is "a residential care institution which provides only supervisory care services to more than five (5) ambulatory persons unrelated to the manager or owner of such a home." Services that may be provided in supervisory care homes are *". . .accomodation, board and general supervision, including assistance to persons in the self-administration of prescribed medications"* (A.R.S. § 36-401.26).

General supervision is defined by regulation as *"protective oversight including daily awareness of resident functioning and continuous needs and functional level assessment, ability to intervene in a crisis situation, and supervision in self-administration of medication."*

When supervising the self-

administration of medications, staff of a licensed supervisory care home may assist residents by reminding them of times of self-administration, reading directions for self-administration, and physically assisting them to self-administer medications. Such physical assistance allows the staff member to offer help to the resident by opening medication containers, for example, but does not permit facility staff to administer medications. In other words, health-realted services, not nursing services, may be provided to residents of supervisory care homes.

An individual living in a supervisory care home should be independent in ambulation with or without support such as a cane or other ambulatory device. The resident must be mentally and physically capable of finding a way to safety without assistance under emergency conditions.

A supervisory care home resident should also be independent in eating, bathing, dressing, and should initiate self-care. While this person may be forgetful or exhibit minor judgment defects, he should usually be in contact with reality and capable of socialization and communication. As is expected of residents in boarding homes and other independent living environments, the supervisory care resident must be continent or responsible for self-care of an indewelling urinary catheter or colostomy.

Personal Care Facilities

In January, 1978, the Legislature reclassified personal care facilities from residential care institutions to a subclass of nursing care institution which, by definition, "*. . .provide medical services, nursing services, health-related services or supervisory care services*" (A.R.S. § 36-401.20). Health-related services refer to "*. . .assistance with eating, bathing, and dressing for individuals who are in need of a protective environment*" (A.R.S. § 36-401.20).

A personal care facility resident may need minimum assistance with transfer activities but is otherwise independent in ambulation with or without assistive devices or independent in wheelchair. While the resident may need an initial setup, he should be able to eat independently and requires only minimum assistance for transfer to tub or shower and in bathing. In general, the resident should be capable of entering into meaningful conversation although memory or judgment may be impaired and there may be occasional episodes of confusion, depression, withdrawal or agitation. The individual should be continent or may have an indwelling urinary catheter or colostomy. He may also require assistance with administration of medications or treatments.

Intermediate Care Facilities

Intermediate care facilities are a subclass of nursing care institutions. Residents of intermediate care facilities require nursing services and the administration of medications and treatment. An intermediate care resident may also require assistance with transfer from bed, chair, or toilet or require assistance in walking. If confined to a wheelchair, the resident should be able to propel the wheelchair himself. Moderate to maximum assistance may be required by the resident to transfer to or from tub or shower and moderate to maximum assistance may also be required by the resident for the bathing process. When meals are set up, the resident may require intermittent assistance while eating. The resident may exhibit marked memory or judgment defects, or may be frequently confused, depressed, withdrawn, agitated or unable to enter into meaningful conversation. He may not be motivated to initiate or participate in self-care activities. In addition, the resident may be incontinent of bowel or bladder.

Skilled Nursing Facilities

Skilled nursing facilities are a subclass of nursing care institution which provide services to inpatients who need professional nursing care on a continuing daily basis but do not require hospital care or care under the daily direction of a physician (A.C.R.R. R9-10-711.29). These services pertain to the preventative, curative and rehabilitative aspects of nursing care performed or supervised by a registered nurse consistent with the attending physician's plan of care and the patient care plan (A.C.R.R. R9-10-711.30).

A skilled nursing facility resident may be bedfast, unable to stand, walk or transfer and may be incapable of self-propelling a wheelchair. Often, the resident must be fed or may require continuous assistance while eating. Also, the resident may require intravenous therapy or tube feeding. A skilled nursing facility resident is one who usually requires total nursing and personal care including bed baths. There may be severe impairment of memory or judgment with chronic confusion, withdrawal, depression or agitation. The resident may not be capable of entering into meaningful conversation or refuses or resists participation in self-care.

Recovery Homes

The Department of Health Services issues letters-in-lieu of license for alcohol and drug abuse recovery centers. Regulations have not been adopted for this category of health care institution; however, the Department surveys these facilities for compliance with local fire, building and zoning codes. Current regulations for supervisory care homes are used as a guideline for the remainder of the survey.

Concluding Statement

The greatest long term benefit for patients, their families, and the community will result from the use of placement criteria that correspond to the patients' physical, social, mental and emotional needs. If a physician needs assistance in locating the appropriate type of facility, he or she may contact the Bureau of Long Term Care of the Arizona Department of Health Services, (602) 255-1115.

State of the Art

Cardiopulmonary Resuscitation

Paul E. Fenster, M.D.
Gordon A. Ewy, M.D., FACC, FACP

Editor:
Eric H. Prosnitz, M.D.

For two decades, the life-sustaining technique of closed chest cardiac massage has been standardized and taught to millions of health professionals and laymen. The technique was based on the hypothesis that compression of the heart between the sternum and vertebral bodies would raise the pressure in the cardiac chambers, and that the action of the heart valves would direct the flow of blood in the forward direction only. The technique, as currently taught, has been used successfully to temporarily support life in countless victims of cardiac arrest. However, recent evidence indicates that this hypothetical mechanism of closed chest massage is probably not operative in the majority of patients. Recent studies offer a more accurate and complete explanation of the mechanism of external cardiac compression used during cardiopulmonary resuscitation (CPR), and lend insight into methods to improve the efficacy of the technique.

From: University of Arizona Health Sciences Center, Department of Internal Medicine, Tucson, AZ 85724. Reprint requests to Dr. Fenster.

The Mechanism of Effective External Chest Compression

Hemodynamic studies during external chest compression in man demonstrate that the pressures within the atria rise almost to the same level as the ventricular pressure.[1] There is, therefore, little or no pressure gradient for forward blood flow within the heart. Cineangiographic and hemodynamic recordings during closed chest cardiac massage in dogs with induced ventricular fibrillation demonstrate that ventricular volumes do not change during chest compression.[2] These same studies show that there *is* gross incompetence of the cardiac valves. These findings indicate that ventricular compression is not the mechanism producing blood flow during external cardiac compression. How, then does the technique increase arterial pressure and perfuse the vital organs?

Niemann, et al noted that in dogs, the initial outflow of blood from the aorta precedes the opening of the aortic valve.[2] They concluded that initial blood flow is therefore produced by *aortic* compression, due to an increase in intrathoracic pressure. Continued flow is produced as the entire thorax functions as a pump; the gradient for flow is from intrathoracic to extrathoracic organs during compressions. The heart is merely a passive conduit.

A rise in intrathoracic pressure may account for blood flow out of the thorax, but does not account for the forward direction of blood flow. The forward blood flow is due to the pressure difference between arterial and venous vessels. For instance, in the cerebral circulation, this pressure differential is maintained by the closure of valves in the internal jugular veins. These valves, usually bicuspid, are located approximately 0.4 cm from the termination of the internal jugular veins.[3] The valves close when intrathoracic pressure is rapidly increased, as by chest compression or by a cough, but do not close with more gradual increases in venous pressures as with heart failure and/or tricuspid regurgitation. The maintenance of a gradient below the diaphragm is more difficult to explain, but is probably due to a "valving" effect of compression on the inferior vena cava.

If the thorax is the "pump" during external cardiac compression then measures which further increase intrathoracic pressure would be expected to augment cardiac output. This has, in fact, been demonstrated. Niemann, et al found that in dogs, chest compression during lung inflation produced significantly greater aortic outflow and aortic pressure than did compression during apnea.[2] The augmentation of pressure and flow occur because the compression force is not dissipated through open airways, but rather enhanced by the positive airway pressure.

Chandra, et al studied the effect of abdominal binding,

another means of raising intrathoracic pressure. An inflatable "girdle device" was used to apply 60 to 110 cm H_2O pressure for 30 second periods. In six victims of cardiac arrest, abdominal binding increased peak systolic pressure from 65 ± 6 to 76 ± 7 mmHg.[4]

Thus, the underlying mechanism of conventional external cardiac compression is the pumping of blood from the thorax, rather than the intracardiac chambers, produced by the rhythmic, external application of pressure. The efficacy of the technique is enhanced by simultaneous application of positive airway pressure and by abdominal binding. These findings explain an earlier report demonstrating that sustained compression, i.e. duration of compression equal to duration of non-compression during external cardiac massage, enhances carotid blood flow. If the entire thorax is the pump, a sustained compression is necessary to allow time for forward blood flow.[5] Faster "jabbing-like" or non-sustained compression is not as effective.

The Lifesaving Cough

The cough is an intrinsic, automatic maneuver which simultaneously applies the elements of successful CPR. A cough is produced by strong contraction of the diaphragm and intercostal muscles, while the glottis is closed. The muscular contraction increases intrathoracic pressure, analogous to external compression with abdominal binding, but to a much greater degree. Coughing produces intrathoracic pressures of 50 to 250 mmHg and occasionally as high as 450 mmHg.[6] The pressure is maintained by the closed glottis, preventing dissipation of the pressure through open airways. The airway contents are explosively discharged when the glottis is reopened, producing an expiration of one to three liters of air. This, coupled with the obligatory post cough inspiration, promotes adequate ventilation in addition to the augmentation of venous return. Repeated coughing should, therefore, be a means of achieving cardiac and pulmonary support in an arrested, but still conscious, victim.

The efficacy of cough CPR in the adult is documented in several case reports. Criley, et al reported eight cases of ventricular fibrillation during cardiac catheterization. In three patients, consciousness was maintained for over a minute by forceful coughing every one to three seconds. The mean systolic pressure during coughs was 139 ± 4 mmHg, significantly higher than the 60 ± 5 mmHg achieved by external cardiac massage.[7] In a subsequent case, consciousness was maintained for 92 seconds.[8]

Cough CPR has also been noted to induce QRS complexes.[8] In one reported case, one cough produced two arterial pulses - one from the cough itself, the second from the cough induced QRS. The electrical activity may result from the mechanical energy from the cough in a manner similar to a chest thump.[9] This depolarization may account for the reported cough-facilitated conversion of ventricular tachycardia.[10]

Clinical Implications

Although the values and limitations of cough CPR are not fully known, it is recommended that patients with the recent onset of ventricular fibrillation or ventricular tachycardia who are still conscious be instructed to cough forcefully and repeatedly. Preparations for definitive treatment should proceed, but the cardiac rhythm should be rechecked prior to administering electric shock if the patient has maintained consciousness. Repeated coughing may also maintain blood pressure in the patient with asystole or bradycardia with hypotension. Rhythmic coughing on demand is an embarrassingly simple technique that may "buy" time by maintaining consciousness of the patient while preparations are made for definitive therapy.

Cough CPR has several potential advantages over external massage. It is simple, easy to perform in any position, and does not require a firm surface. The trauma sometimes associated with external compression, including rib fractures, liver laceration, and cardiac contusion, are avoided. Cough CPR is instantly available and may permit a single rescuer to prepare for defibrillation without delay. Finally, the coughs insure that breathing is maintained.

Further studies are needed to determine the optimal technique of cardiopulmonary resuscitation. It would appear that the application of lung inflation simultaneously with chest compression produces better perfusion than does lung inflation between compressions. The optimal frequency of inflations, the inflating pressure, the duration of lung inflation all need to be better defined. The role of abdominal binding, as well as the risks of this procedure, are also incompletely known. We may expect, however, that as more information is obtained, the present "standards" and the currently recommended technique of CPR will be changed.

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Arizona Medicine

Editorial

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Walk-In Clinics: A Vital Component of Education and Service at the Arizona Health Sciences Center

One tends to identify academic medicine with its research mission, be it esoteric projects that expand the frontiers of medical knowledge or innovative educational programs that train the new generation to thrive in the ever increasing complexity of medical practice. More mundane perhaps, but no less vital to the educational mission, is the need to develop models of effective patient care services. The patient care system at the University of Arizona Health Sciences Center (AHSC) is the site where these diverse missions come together.

At the AHSC Clinics, the general public is served through patient care programs that cover the full spectrum of medical specialties and services. For the fiscal year, ending June 30, 1979, over 136,000 patient visits were recorded in forty-eight distinct clinics. Providing these services are 220 clinical faculty members along with 180 house officers at various levels of postgraduate training. The full-time clinical faculty are only engaged in direct patient care on a part-time basis because of their medical education responsibilities. While this bit of information is far from being earth shaking, the part-time aspects of the faculty member's role require significant accommodations that must be made in the interests of good patient care.

Patients have a variety of reasons for utilizing the services provided in the AHSC Clinics. Access to a physician on an urgent basis and continuity of care are certainly high on the list of patient expectations. Given the highly structured nature of clinical teaching programs, providing easy access for undiagnosed problems is a remarkably difficult task. The fact that a patient's personal physician at the AHSC may not always be available for continuity requires that an effective alternative be provided. Resolving this problem in a manner that enhances the educational experience adds a further challenge.

The Department of Internal Medicine has devised a program that meets the requirements of education and service in a very harmonious fashion. The department is strongly oriented toward specialty programs that are designed to provide longitudinal care to patients with specific diseases. The burden of providing care to patients who do not fit into the specialty clinic model is the responsibility of the Section of General Internal Medicine. Patients are seen and followed on an appointment basis by teams of general internists and housestaff that are assigned by the day of the week. This system works well for new patients with non-urgent problems. A separate program, the Walk-In Clinic, was established in 1977 to meet the requirements of patients who feel that they need to be seen without delay. This Clinic is open five days a week and staffed through a one month rotation by a second year house officer, under the supervision of a general internal medicine faculty member. Each day, approximately eight to ten patients are seen with episodic problems that present a wide spectrum of complexity. About one-half of these patients are new to the Health Sciences Center. The remainder are primarily ongoing patients with an episodic acute problem. From the perspective of the house officer, this program is popular. First, it provides an opportunity to gain experience in patient management similar to that expected in actual practice. Second, this point-of-first-contact experience provides an opportunity to evaluate previously undiagnosed problems not usually found in the specialty clinics.

Although this discussion focuses on activities within the Department of Internal Medicine, similar programs have been developed to provide urgent care in other medical disciplines as well. From an institutional standpoint, the Walk-In Clinic model adds a dimension of service that fills a major gap and provides a measure of responsiveness to the public we serve while meeting an important educational need.

Paul W. Joy
Administrator, Medical Service Plan

Louis J. Kettel, M.D.
Dean, College of Medicine

Correspondence

Editor—

It has been believed since antiquity that if our children asked us for bread we would not give them a stone, and if they asked us for a fish we would not hand them a serpent. That is about to changed! What we are doing to succeeding generations is not only immoral, it's beyond belief.

Our own kind, in future lifetimes will bear the burden of our profligate, chuckle-headed social security scheme while we tap the grossly underfunded program now. This con game is made worse because even though they see through it, they have no option to reject it.

That isn't all. Because of inflation in our time, and our bureaucratic regulatory agencies that ignore inflation, future consumers of power for example, will have loaded upon them astronomic costs for replacement of utilities that we are wearing out. The Con Edison Report documents the cost of their Indian Point Nuclear Generating Plant No. 2, as $215 million in 1973. It is estimated that it will cost $5 billion to replace it in 2006, (give or take a few years). The regulatory agencies in controlling current rates, allows the company to charge as depreciation only the recovery of $215 million by 2006. One of two things has got to happen; either the next generation comes up with the difference between $215 million and $5 billion in order to maintain what we now have, or they burn buffalo chips. The problem is we don't have any more buffalo chips either.

Paul B. Jarrett, M.D.
Phoenix, Arizona

Editor—

Re: Review article on Stage I Cancer of the Endometrium by Earl A. Surwit, M.D. - January 1980.

The article by Dr. Surwit was certainly quite thorough and worthwhile. However, I would like to clarify one issue regarding the treatment of this complex disease. It must first be noted that there seems to be two types of endometrial cancer; that which is associated with estrogen administration which behaves in a benign fashion; and that which arises denovo from other exogenous and endogenous etiologies.

I have reviewed over 100 cases of Stage I endometrial carcinoma treated by numerous physicians in private practice. The overwhelming majority of these patients had been on Estrogens and almost all were well differentiated tumors that had either no or minimal superficial invasion of the myometrium. There was no evidence of vaginal cuff recurrence in any of these patients although none received post or pre-operative radiation. Therefore, in this group of patients, total abdominal hysterectomy and salpingo oopherectomy appears to be the treatment of choice without need for further therapy. Therefore, the treatment for patients with this disease needs to be individually tailored to each patient and the statment that "post-operative vaginal radiation is required for *all* patients" is not tenable.

Sincerely,

Kent J. Rossman, M.D.
Phoenix, Arizona

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In Memoriam

Albert Bodaski, M.D.
1904-1980

Albert Bodaski, M.D., was born in Youngstown, Ohio, January 23, 1904, and was graduated from the University of Minnesota College of Medicine 1938.

I first met Al at Camp Young, California, July 1942, a grim camp of Army squad tents in the vast forbidding desert between Blythe and Indio where mechanized divisions were being trained for North African warfare. We were Army Medical Corps initiating the 5th Field Hospital to go in with invasion troops. Like a lot of young doctors he left his home, family, and friends, because the war could not be won without him.

Our next four years were spent in New Guinea, and island hopping in the Pacific. As with service people in the Pacific, hopes of coming back to his beloved family in Minnesota sustained him through the misery, wretchedness, hopelessness, jungle and war stench. In the midst of so much chaos and viscious warfare, Al not only ministered to battle casualties, but involved himself in bucking up the emotionally overwhelmed, the futile, the depressed, the agitated, the isolated, the honored and the dishonored. He was decent, friendly, understanding, and always a source of encouragement, and support.

At war's end, he made it back to his beloved stamping ground in Tyler, Minnesota, and his beautiful family. I didn't see him again until 1970 when he came down here to Phoenix to convalesce in our desert sunshine subsequent to a cervical laminectomy from an accident sustained in a Minnesota blizzard. I'm sure he was a joy and delight in serving his Minnesota population as doctor, friend and confidante.

Until four years ago, Al tried being active in the Phoenix private practice community, keeping his practice low key because of his health problems, which must have been emotionally very disturbing to him. There were several C.V.A.'s during the last couple of years, the last on May 30, 1980, taking its final toll.

His R.N. wife, Gunver, his Tucson Lutheran minister son, Albert Jr. and his Prescott community college teacher daughter, Barbara Gardner, are most grateful for the wonderful care and service rendered him by his medical friends and colleagues, and the nursing staff at St. Joseph's Hospital.

A fine soldier, wonderful guy, and doctor leaves us for his Valhalla. Bon Voyage, Al.

Samuel I. Rothman, M.D.

ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

MATERNAL AND CHILD HEALTH CARE COMMITTEE

The meeting of the Maternal and Child Health Care Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, May 24, 1980, convened at 10:14 a.m., William C. Scott, M.D., Chairman, presiding.

Statewide Perinatal Mortality Review Program

The committee discussed at great length the need for a statewide perinatal mortality review program and the steps which have already begun to be taken on a county level. Additionally, Dr. John Kelly advised the members that there was, in the mail, a proposal for the initial development of such a review program and, once received, it could be discussed and considered in greater depth.

It was moved and carried that an Ad Hoc Committee on Perinatal Mortaility be established that would ultimately become a permanent subcomittee.

Advisory Task Foce on Maternity Care

Emily Jenkins-Reed, J.D., Director of the Arizona Family Planning Council and member of the Advisory Task Force on Maternity Care, discussed briefly with the committee the Summary Report prepared by that group and submitted to the membership for its review, consideration and information.

The committee then reviewed, item by item, the recommendations of the Task Force and responded to each by comment of concurrence, objection or modification, as more specifically set forth in Exhibit "A" attached hereto and by reference incoporated herein. (*These comments are available at the ArMA Offices and will not be printed here.*)

It is the desire and intent of the committe that such comments be submitted in written form to the Advisory Task Force for its consideration prior to implementation of any of its recommendations.

Additionally, in reviewing Recommendation No. 2.9, relating to the establishment of back-up service within the state, the committee took the following action.

It was moved and carried that the Maternal and Child Health Care Committee recommend to ArMA's Legislative Committee that it consider and explore with the Legislature the drafting and passing of a law to establish back-up medical services on a statewide basis.

The committee, in dealing with Recommendation No. 4.7, determined that in one of the upcoming issues of the Association's newsletter *Medical Memos*, mention should be made to the Task Force's findings that a closer relationship between providers and family during the prenatal period would lend itself toward greater insight as to the family's needs after the infant's birth and "pediatricians or primary care providers are encouraged to become involved with the family during the prenatal period."

AMA House of Delegates Resolution No. 22

The committee received and reviewed Resolution No. 22, "Out-of Hospital Birth Risks," adopted by the AMA House of Delegates during its July 1979 Annual Meeting and it was determined that the same was of sufficient import to disseminate to the general membership via *Medical Memos*.

Prevention of Mental Retardation

The committee received and reviewed a letter dated February 29, 1980 from Valley of the Sun School, regarding general input on the implementation of an Arizona program for prevention of mental retardation. It was consensus of the membership that Dr. Scott, as Chairman of the committee, would handle any further contacts regarding this matter.

Arizona Perinatal Program/Arizona Perinatal Trust

Dr. Harold Gabel, Executive Director of the Arizona Perinatal Program, updated the committee on the status of APP's proposal to the Robert Wood Johnson Foundation for the creation of the Arizona Perinatal Trust and presented for the committee's consideration a request being made to the Association for in-kind support. Following extensive discussion,

It was moved and carried that the Maternal and Child Health Care Committee recommend to the Board of Directors that it approve, after the July 31, 1980 termination of the Arizona Perinatal Program and if, in fact, the Arizona Perinatal Trust is established and comes into existence, in-kind support to the Arizona Perinatal Trust by way of continuation of services presently being purchased or equipment being used by the Arizona Perinatal program at no cost or expense to the Arizona Perinatal Trust for a period of three years from the date of inception of such trust.

The committee felt that, when it was determined when this matter would be placed on the agenda of a Board of Directors meeting, it would be appropriate for Dr. Gabel to be invited to attend such meeting to speak on behalf of the request and answer any questions which might arise.

Reporting of Out-Of-Hospital Complications

Dr. William Carlile verbally reported to the committee regarding efforts to impose reporting of out-of-hospital complications and advised that information and data was being complied as to numbers and types of such complications throughout the state and, when available, would be distributed to the committee for its review.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, May 30, 1980, a quorum being present, convened at 6:47 p.m., John T. Clymer, M.D., President and Chairman, presiding.

Arizona Health Service Corp.

Mr. Robinson updated the committee on the status of the Arizona Health Service Corp. contract, advising that the Association had finally received the balance due thereunder and that the matter was finally closed.

Medical Economics Committee Negotiating Arm for Physicians

Dr. Kurtz, as Chairman of the Medical Economics Committee, reviewed briefly for the committee an article recently published in *Medical Leadership News*, a newsletter of the Union of American Physicians and Dentists, relating to conflicts which have occured in the State of California by reason of the California Medical Association's attempt to negotiate on behalf of its physicians. Dr. Kurtz advised that his committee would take the matter under further consideration and study at its next meeting in an effort to insure that the committee and the Association would not be placed in a similar type situation by their representation of member physicians.

BOMEX Appointments

The committee again considered the earlier request of Governor Babbitt for submission of recommmendations for appointment to the Board of Medical examiners to fill a vacancy which would occur on June 30, 1980, with the expiration of the term of George Scharf, M.D. togethter with recommendations submitted by the Maricopa County Medical Society on May 14, 1980.

It was moved and carried that the Arizona Medical Association submit the names of Richard D. Zonis, M.D. and Paul B. Jarrett, M.D. to Governor Babbitt as its recommendations for candidates for appointment to the Board of Medical Examiners to fill the vacancy which would occur on that Board on June 30, 1980, by reason of the expiration of term of office of George Scharf, M.D.

Optometric Practice Act Senate Bill 1015

The committee discussed at great length the recent passage of the Optometric Practice Act - Senate Bill 1015 -by the Arizona Legislature and the provisions therein contained and it was determined that Dr. Clymer would contact Dr. Thomas Moore, as the representative of the Arizona Ophthalmological Society, requesting his suggestions of ophthalmologists who might be considered by the Association as recommendations for appointment to the State Board of Optometry. Additionally, Dr. Moore would be requested to be present at the next meeting of the Executive Committee scheduled for June 27, 1980, to discuss with the committee how ArMA might best assist in the implementation of the other provisions of this newly-enacted legislation.

Arizona Perinatal Program

The committee reviewed the letter request of the Arizona Perinatal Program dated May 16, 1980, wherein in-kind support was sought, should the Arizona Perinatal Trust be established upon the termination of the Program on July 31, 1980, together with the recent action taken by the Maternal and Child Health Care Committee of the Association relating to the like request.

It was moved and carried to refer the request of the Arizona Perinatal Program for in-kind support to the Finance Committee, with the Executive Committee's recommendation that the Finance Committee consider support of the Arizona Perinatal Trust, if established, for a period of three years, limted, however, to the following: continued provision of accounting services and transfer of title to certain equipment presently being used by the Arizona Perinatal Program and standing in the name of the Arizona Medical Association Foundation.

ArMA Resolution 1-80

It was determined that discussion of Resolution 1-80 relating to Unified Membership Between the County Medical Societies and the Arizona Medical association passed by the House of Delegates at its recent annual meeting and any further action thereon would be placed on the agenda of the

Executive Committee for its meeting on June 27, 1980.

Long Range Planning Committee

Dr. Clymer advised the committee that Dr. John E. Oakley had accepted chairmanship of the newly-established Long Range Planning Committee but had deferred appointment of the membership until further determinations could be made as to the exact goals of the committee.

During the discussion on this matter, however, it was the feeling of the membership that consideration should be given to a bylaws amendment which would change the present requirment for a two-thirds majority vote in the House of Delegates. The committee was advised that there were several options available in order to accomplish this and Mr. Jacobson, as legal counsel for the Association, was requested to prepare his opinions and recommendations relative thereto for consideration at a future meeting.

Indemnity Insurance

The opinion of Snell & Wilmer dated May 6, 1980 pertaining to insurance indemnifying officers, directors and employees of nonprofit corporations was reviewed and, following a lengthy discussion,

It was moved and carried that Bruce E. Robinson, Executive Director of the Association, should immediately proceed to obtain bids for policies of insurance that would fund the indemification of the Association's officers, directors and employees allowed by law for consideration and recommendation to the Board of Directors.

BOMEX - Rule 4-16-17

The recent Notice of Proposed adoption of Rules, i.e., R4-16-17 (Fraud; Definition), issued by the Board of Medical Examiners, on which hearing is scheduled for June 5, 1980, was the subject of lengthy discussion by the committee and the Association's legal counsel. It was determined that Dr. Kurtz would immediately contact the BOMEX Chairman of the Board to determine, if possible, the reasoning behind the proposed rule and whether the Board might be amenable to postponing the present hearing date in an effort to timely study the wording contained in the rule as noticed. It was further determined that Dr. Clymer, as President of the Association, would appear, with legal counsel, at the hearing if held as scheduled, to set forth the Associations' position that, while it was not objecting to a rule which would prohibit the charging of "grossly excessive fees," it felt that further consideration should be given to the wording of such a rule and that an extension of time was required prior to any final action of the Board in order that legal counsel might arrive at some mutually agreeable terminology.

Uniform Health Care Study Committe

The committee considered a request by the Arizona Community Action Association, Inc. relative to the establishment of a committee to study health care delivery systems to the poor in Arizona. Since Dr. Crisp has been involved in past discussion with members of the group forming the Uniform Health Care Study Committee,

It was moved and carried to recommend to the Arizona Community Action Association, Inc. that William E. Crisp, M.D. be appointed to the newly-formed Uniform Health Care Study Committee which would review the health care delivery systems to the poor in the state of Arizona.

Arizona Atomic Energy Commission

A letter from the Arizona Nuclear Medicine Physicians regarding the reorganization of the Arizona Atomic Energy Commission into the Radiation Regulatory Agency and seeking support of the appointment of a Phoenix health physicist, Martin T. J. Hilger, as a member of that agency was considered by the committee.

It was moved and carried that the Arizona Medical Association transmit by letter to Governor Babbitt its concurrence in the recommendaiton that Martin T. J. Hilger of Phoenix be appointed to serve on the recently-established radiation regulatory agency/

1981 Annual Meeting Dates

Mr. Robinson reminded the committee that the Board of Directors had recently taken action to change the annual meeting dates of the Association to coincide with the "off-season" of area hotels; and that, due to the fact that 1981 had already been committed for, no changes were made in those dates. However, he additionally advised that The Pointe Resort, the hotel hosting the 1981 Annual Meeting, had indicated a willingness to alow the dates to be changed from April to the latter part of May, enabling a better room rental rate to the attending physicians, and due to the need to give the hotel an immediate positive commitment Mr. Robinson required direction from the Executive Committee.

It was moved and carried to allow the Association's Executive Director to confirm with The Pointe Resort a change of the annual meeting dates for the Association's annual meeting from April 22-26, 1981, to May 26-30, 1981.

Nuclear Emergencies

Dr. Baker addressed the committee regarding Aeizona's formal plan for handling of nuclear emergencies and requested concurrence from the committee to his continuing discussion with those involved, as well as with Willard R. VanNostrand, M.D., Chairman of the Sections on Emergency Care and Disaster Medicine, established under ArMA's Professional Committee, to determine what role ArMA might play in this overall planning. Concurrence was given by the entire committee, with Dr. Baker to report back following such discussions.

ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE

The meeting of the Articles of Incorporation and Bylaws Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, May 31, 1980, convened at 10:10 a.m., Charles E. Henderson, M.D., Chairman, presiding.

Reorganization of House Of Delegates

The committee reviewed and discussed the charge to it by the Board of Directors that it consider the bylaws change that would be required to reorganize the ArMA House of Delegates to include specialty society participation. In addition, it reviewed information from the states of Texas, Pennsylvania and California, wherein this type of activity had already occurred, as well as the advice from Dr. Scott that, pursuant to a survey he made of the specialty societies within the state, it would appear that most, if not all, were in favor of and indicated interest in such type of reorganization.

Following a very involved and lengthy discussion, the committee determined that it favored inclusion of specialty society participation in the House of Delegates and proceeded to set down criteria by which a specialty society would be affored the privilege of having delegate representation.

At this point, the committee determined that the Chairman, together with staff, would prepare, in proposed form, an amendment of the Bylaws, Chapter VIII, House of Delegates, which would effectuate the intended reorganization (see Exhibit "A" attached here to and by reference incorporated herein). Additionally, the committee felt a determination of exact numbers of each specialty society within the state was necessary, and that, when available, the committee would review both documents prior to formal recommendation to the Board of Directors.

Intern and Resident Dues Bylaws Amendment

The committee considered the action taken by the House of Delegates in April of this year by the addition of an amendment to Resolution 2-80 providing that ArMA dues for interns and residents be waived for their first year of membership and reviewed the applicable portions of the Bylaws, taking the following action.

It was moved and carried to recommend to the Board of Directors that, in order to accomplish the action taken by the House of Delegates in April of this year by its amendment to Resolution 2-80 providing that ArMA dues for interns and residents be waived for their first year of membership, the following changes in the bylaws be made.

That Chapter II, Section 3(A) Active Members, subsection **Rights** be amended to read as follows:

"Rights. An active member shall have all the rights and privileges of the Association as herein provided. Intern and Resident active members shall pay NO DUES FOR THE FULL CALENDAR YEAR IN WHICH THEY FIRST BECOME A MEMBER AND, THEREAFTER, shall pay ten per cent of the dues of other active members and will be exempted from special assessments."

In revising Chapter IX, Section 5. Exemptions, to bring it into conformity with the foregoing amendment, the committee determined that student members had inadvertantly been omitted from subsection (a) and (b) of Section 5, Chapter IX, and therefore determined that Chapter IX, Section 5. Exemptions, subsections (a) and (b) be amended to read in their entirety as follows:

"(a) Members exempt from the payment of dues shall include (1) 50 year club, (2) Associate Members, (3) Affiliate Members, (and) (4) Honorary Members, AND (5) STUDENT MEMBERS. Members 70 years of age or older, and other members by reason of physical disability, illness or financial hardship may also be exempted. A member must request such exemption, and secure recommendation by his county medical society and be approved therefor by the Board.

(b) Members exempt from the payment of special assessments include (1) 50 year club, (2) Associate Members, (3) Affiliate Members, (and) (4) Honorary Members, (5) INTERN AND RESIDENT ACTIVE MEMBERS, AND (6) STUDENT MEMBERS. Members 70 years of age or older, and other members by reason of physical disability, illness, or financial hardship may also be exempted. A member must request such exemption, and be approved therefor by the Board."

Exhibit "A"
Proposed Amendment to the Bylaws of The Arizona Medical Association, Inc.

It was moved and carried by the Articles of Incorporation and Bylaws Committee to recommend to the Board of Directors that, in order to effectuate the reorganization of ArMA's House of Delegates to include specialty society participation, Chapter VIII, House of Delegates, of the Bylaws of the Arizona Medicial Association, Inc. be amended as follows:

SECTION 1. COMPOSITION OF HOUSE; MEETING, SHALL BE REWRITTEN TO READ IN ITS ENTIRETY AS FOLLOWS:

"Section 1. Composition of House; Meeting: — The House shall constitute the voting body of the Association and shall be composed of the elected Delegates of the county societies, THE SPECIALTY SOCIETIES, elected Delegates of each duly constituted chapter of the American Medical Student Association in Arizona, elected Delegates of the duly constituted chapter of the Arizona Medical Association Housestaff Section, and members of the Board. Delegates who are thereafater elected as officers do not by such election lose their status as voting Delegates in the House. The past presidents of the Association shall be ex-officio members of the House of Delegates without the right to vote.

The House shall meet at least once a year at the time of the Annual Meeting. In addition, special meetings of the House may be held at any time, upon at least six weeks' notice thereof to the Delegates, at the call of the board, or upon the call of twenty Delegates representing at least eight county societies. THE SPECIALTY SOCIETIES, THE ARIZONA MEDICAL ASSOCIATION HOUSESTAFF SECTION AND EACH DULY CONSTITUTED CHAPTER OF THE AMERICAN MEDICAL STUDENT ASSOCIATION."

SECTION 2. NUMBER OF DELEGATES, SHALL BE REWRITTEN TO READ IN ITS ENTIRETY AS FOLLOWS:

"Section 2. Number of Delegates: —

(A) COUNTY SOCIETIES. THOSE SPECIALTY SOCIETIES HAVING A NATIONAL BOARD RECOGNIZED BY THE AMERICAN BOARD OF MEDICAL SPECIALTIES AND HAVING A STATE ORGANIZATION OR STRUCTURE EXHIBITING A SLATE OF PERIODICALLY ELECTED OFFICERS, AN ESTABLISHED CONSTITUTION AND BYLAWS, AND A FREQENCY OF MEETINGS OF AT LEAST ONCE YEARLY, AND A MINMUM OF TWENTY MEMBERS PRACTICING IN ARIZONA, A MAJORITY OF WHOM ARE ALSO MEMBERS OF THE ASSOCIATION, SHALL BE ENTITLED TO REPRESENTATION IN THE HOUSE BY ONE DELEGATE, OR HIS ALTERNATE, BOTH OF WHOM MUST BE MEMBERS OF THE ASSOCIATION, AND ONE ADDITIONAL DELEGATE, ALSO A MEMBER OF THE ASSOCIATION, FOR EACH 100 ACTIVE MEMBERS AS DETERMINED ON OCTOBER FIRST PRECEEDING THE ANNUAL MEETING.

WHEN A MEDICAL SPECIALTY REPRRESENTED BY A NATIONAL BOARD HAS MORE THAN ONE QUALIFYING SPECIALTY SOCIETY WITHIN THE STATE, THIS SECTION SHALL BE DEEMED TO APPLY TO THAT SOCIETY WHICH, IN THE OPINION OF THE BOARD OF DIRECTORS OF THIS ASSOCIATION, IS THE MAJOR VOICE FOR THAT SPECIALTY IN STATE AND LOCAL AFFAIRS AFFECTING THAT SPECIALTY.

(C) HOUSESTAFF SECTION: STUDENT ASSOCIATION: Each duly constituted chapter of the Arizona Medical Association Housestaff Section and each duly constituted chapter of the American Medical Student Association in Arizona shall each be entitled to representation in the House by two Delegates or their alternates."

SECTION 3. ELECTION OF DELEGATES: LIST THEREOF, SHALL BE REWRITTEN TO READ IN ITS ENTIRETY AS FOLLOWS:

"Section 3. Election of Delegates; List Thereof: — Sufficiently in advance of the Annual Meeting each county society, EACH SPECIALTY SOCIETY, The Arizona Medical Association Housestaff Section, and each duly constituted chapter of the American Meidcal Student Association in Arizona shall elect Delegates and an equal number of alternates to represent it in the House. In the absence of any Delegate, his alternate may vote in his name on any question before the house. No alternate shall be eligible for election to office, but he may be appointed to membership on House committees in the absence of the Delegate. The secretary of each county society, EACH SPECIALTY SOCIETY, (and of) the Arizona Medical Association Housestaff Section and each chapter of the American Medical Student Association shall send the list of such elected Delegates and alternates to the Secretary of the Association not later than two months before the Annual Meeting. Representation in the House shall be contingent upon compliance with this provision. A member to be seated must present evidence at the time of his appearance at the House(,) of his official election by his county society, HIS SPECIALTY SOCIETY, (or) the Arizona Medical Association Housestaff Section, or chapter of the American Medical Student Association."

SECTION 7. QUORUM: MINIMUM REQUIREMENTS FOR VOTE, SHALL BE REWRITTEN TO READ IN ITS ENTIRETY AS FOLLOWS:

"Section 7. Quorum; Minimum Requirements for Vote: — Fifteen Delegates, or their alternates, representing seven county societies, SPECIALTY SOCIETIES, THE ARIZONA MEDICAL ASSOCIATION HOUSESTAFF SECTION OR EACH CHAPTER OF THE AMERICAN MEDICAL STUDENT ASSOCIATION, shall constitute a quorum, and unless otherwise specified herein, a majority vote of the quorum shall be required for action."

Comings & Goings

The Barrow Neurological Institute of St. Joseph's Hospital and Medical Center in Phoenix, Arizona, announces the appointment of Robert M. Crowell, M.D., currently Associate Professor of Surgery (Neurosurgery) of Harvard Medical School and Associate Visiting Neurosurgeon of the Massachusetts General Hospital, as Chairman of the Division of Neurological Surgery, Director of the Neurosurgical Residency Program and of the Stroke Research Laboratory in this institution as of September 1, 1980. He will succeed John R. Green, M.D, who has been the Chairman of the Division and the Residency Program since it was founded and who will continue with his appointment as Director of the Institute, with neurosurgical practice, teaching and clinical research privileges.

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

The Third International Conference on the Adjuvant Therapy of Cancer will be held at the Tucson Convention Center, Wednesday, March 18 through Saturday, March 21, 1981, under the sponsorship of the Cancer Center Division of the University of Arizona. Drs. Sydney E. Salmon and Stephen E. Jones will serve as Conference Co-Chairmen. Abstract forms are now available; the deadline for submisison of abstracts (prepared in the format of AACR/ASCO) is November 1, 1980. For abstract forms or futher information, please contact: Mary Humphrey, Conference Coordinator, Cancer Center Division, University of Arizona, Tucson, Arizona 85724.

AUGUST

Arizona Perinatal Medicine Program
August 8 & 9, Yavapai Community College, Prescott, Arizona. Sponsor: Arizona Perinatal Program, 801 W. Bethany Home Rd., Phoenix, AZ. Contact: Arizona Perinatal Program Offices. Approved for 12 hours of category 1 credit.

Pediatric Developmental Diagnosis
August 14-16. U of A Health Sciences Center: Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith. Office of Continuing Medical Education & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Symposia de Santa Fe
August 21-23. Santa Fe Hilton, Santa Fe, New Mexico. Sponsor: Symposia de Santa Fe. Contact: W. J. Levy, M.D., Sec.-Treasurer, P.O. Box 5175, Coronado Station, Santa Fe, New Mexico 87502.

Advanced Cardiac Life Support Program (Provider Course)
August 22-23. Arizona Medical Assn. Offices. Sponsor: A.C.E.P., Arizona Affiliate, American Heart Assn & ArMA. Contact: Carol Turner, Arizona Medical Assn., 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of credit.

SEPTEMBER

Advanced Cardiac Life Support Program (Instructor)
September 5-6. Arizona Medical Assn. Office. Sponsor: A.C.E.P., Arizona Affiliate, American Heart Assn. & ArMA. Contact: Carol Turner, Arizona Medical Assn., 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Current Psychotherapeutic Perspective in Working with Children & Adolescents
September 6-7. Registry Hotel, Scottsdale, Az. Sponsor: Professional Seminars of California. Contact: F. O. Webley, MSW, Professional Seminars of California, 6850 Brockton Ave. #107, Riverside, CA 92516. Approved for 12 hours of category 1 credit.

Personal Instruction Course In Intra & Extra Capsular Cataract Extraction with IOL Implantation.
September 10-13 and 24-27, Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006.

Pediatrics
September 13. Arizona Health Sciences Center, Tucson. Sponsor: U of A College of Medicine.Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

The Determination of Toxicity of Chemical Products.
September 16-18. Plaza International, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center of Occupational Safely and Health, U of A Health Scineces Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Dean's Clinical Rounds
September 18. U of A Helath Sciences Center. Sponsor: U of A College of Medicine. Contact: Lolly Dawn, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Evaluation of Peripheral Neuropathy
September 24. Boswell Hosp. Rm. C119, Sun City. Sponsor and Contact: Martha R. Newby, Dir. of Education, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372.

Comparative Techniques of Weight Control
September 26-27, Scottsdale Hilton Hotel, Scottsdale, AZ Sponsor: University of Arizona College of Medicine, and the Central Arizona District Dietetic Association. Contact: Janet Price, R.D., CADDA, 1051 S. Dobson Rd., No. 56, Mesa, AZ 85202. Approved for 10 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
September 26-27. Arizona Medical Assn. Offices. Sponsor: A.C.E.P., Arizona Affiliate, American Heart Assn., & ArMA. Contact: Carol Tuner, Arizona Medical Assn., 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

OCTOBER

Personal Instruction Course In Intra & Extra Capsular Cataract Extraction with IOL Implantation
October 8-11 and 22-25. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006.

Clinical Seminar Series "Pulmonary Diseases"
October 11. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724.

Management of Common Neurologic & Neurosurgical Problems
October 17-18. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucosn, AZ 85724. Approved for hour per hour category 1 credit.

Welding Safey and Health for the Mineral and Construction Industry
October 24. Marriott Hotel, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizoan Center for Occupation Safety and Health, U of A Helath Sciences Center, Tucosn, AZ 85724. Approved for hour per hour category 1 credit.

NOVEMBER

Personal Instruction Course In Intra & Extra Capsular Cataract Extraction with IOL Implantation
November 5-8 and 19-22. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 19th Street, Ste 105, Phoenix, AZ 85006.

Clinical Seminar Series "Endocrinology"
November 8. U of A health Sciences Center, Tuson. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Medical Society of the United States and Mexico
November 12-15, Guadalajara, Jalisco, Mexico. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Exec. Secy., 3161 N. Pantano Road, Tucson, AZ 85715.

Dean's Clinical Rounds
November 13. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Lolly Dwan, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Arizona Pediatric Society
November 14-16. Westward Look Resort, Tucson. Sponsor: U of A College of Medicine & Arizona Pediatric Society. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

"Risk Managment ofr Safety Professionals"
November 17. Student Union-U of A, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center for Occupational Safety and Health, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Geriatrics Conference
November 20-21. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Chrity Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Comprehensive Review in Toxicology
November 20-22. Sheraton-Pueblo Inn, Tucson. Sponsor: Denver Institute of Clinical Toxicology. Contact: CRIT, c/o Peter Bryson, M.D, Emergency Department, Denver General Hospital, 8th Ave. & Cherokee St. Denver, CO 80204. Approved for 15 hours of category 1 credit.

DECEMBER

2nd Annual Sports Medicine Symposium
December 3-6. Marriott Hotel, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

International Congress on Ericksonia Approaches to Hypnosis and Psychotherapy.
December 4-8, Phoenix, Arizona. Sponsor: Maricopa County General Hospital and The Milton H. Erickson Foundation, Inc. Contact: Jeffrey K. Zeig, Ph.D. The Milton H. Erickson Foundation, Inc. 1935 East Aurelius Avenue, Phoenix, Arizona 85020. Approved for hour per hour category 1 credit.

1981

FEBRUARY

Third Annual International Catarct Surgery Symposium
February 16-18, 1981. Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center. Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.

Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff

Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.

Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists

Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.
Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.
Neurology-Neurosurgical
Fridays, 8-9 a.m . First Floor Conference Rm.
Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.
Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.
Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.
Tumor Board
last Thursday of the month, 12 Noon, Educ. Center Classroom I & II, May, July, Sept, & Nov.
Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.
Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.
Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.
Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

EL DORADO HOSPITAL TUCSON, (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.
Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A Hinchcliffe, M.D. Approved for category 1 credit.
Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 A.M., Contact: Paul Bergeson, M.D.
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM & R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.
Attending's Hour
5th Monday, 12:30 P.M., Contact: J. Goldberger, M.D.
Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman
Ophthalmology Conference
1st Monday, 12:30 P.M., Contact: Bruce Gillers, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.
Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: B. Phibbs, M.D.
Clinical Pharmacology Conference
5th Tuesday, 12:30 P.M., Contact: M. Katz, Clinical Pharm.
12Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact J. Laguna, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: M. Baker, M.D.
Medical Surgical Grand Rounds
2nd Wed., 12:30 P.M., Contact: Chief Medical Resident
Medical Conference
1st Wed, 12:30 P.M., Contact: Chief Medical Resident
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: B. Phibbs, M.D.
Endocrine Conference
2nd Thursday, 12:30 P.M., Contact E. Munjack, M.D.
GI Conference
4th Thursday, 12:30 P.M., Contact: C. Kanun, M.D.
Mortality & Morbidity Conference
3rd. Thursday, 12:30 P.M., Contact: E. Munjack, M.D.
OB/GYN Dept. Meeting
2nd Thursday, 2 P.M., Contact J. Wurzel, M.D.
OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: J Wurzel, M.D.
OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 P.M., Contact: J. Wurzel, M.D.
Clinical Pathology
5th Thursday, 12:30 P.M., Contact: J. Fleishman, M.D.
Renal Conference
1st Friday, 12:30 P.M., Contact: T. Pence, M.D.
Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D.
Rheumatology Conference
3rd Friday, 12:30 P.M., Cntact J. Goldberger, M.D.
Chest Conference
5th Friday, 12:30 P.M., Contct: D. Engelsberg, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008
Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 85008. Contact: Leonard Tamsky, M.D.

Medicine Conference
Daily 12-1 P.M., Contact: S. Schaffner, M.D.
Chest Surgery Conference
Weekly, Mondays, 1:30 P.M., Santa Cruz Rm.
Hepatology Conference
2nd Tuesday, 1:00 P.M., Contact: Walt Koppenbrink, M.D.
OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Room
Obstetrical Problem Conference
Weekly Tuesday, 7:30 A.M., Yavapai Rm.
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.,Contact: Robert Ganelin, M.D.
Urology Discharge Planning Conference
Weekly, 11:30 A.M., Station 42
Hand Surgery Conference
Weekly Wed, A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly, Wed. 7:30 A.M., Yavapai Rm.
Clinical Psychiatric Conference
Weekly, Wed., 11-12 P.M. Mental Health Annex, Rm. 1346.
Surgery Conference
Weekly Wed., 7-8 A.M., Surgical Dept.
Cardiology Conference
Weekly Thurs., 2 P.M., Santa Cruz Rm.
OB/GYN Resident Conference
Weekly Thurs., 12 P.M., Yavapai Rm.
GYN Endocrine Seminar
1st & 3rd Friday, 12:30 P.M., Sanata Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & HEALTH CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.
Medical & Surgical Conference
Weekly Monday, Wed. and Friday, 12 Noon
OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conf. Rm.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for Category 1 credit.
Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Contact: D. Randall, M.D.
Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 A.M.
Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 A.M., Conf. Rm. A, Contact: Drs. L. Moher & L. Fairbanks.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for Category 1 credit.
Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Clinical Conferences
Weekly Tuesday, 12 Noon, Kiva Conference Room
Tumor Board Conference
Weekly Friday, 12 P.M., Kiva Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.
Family Practice Conference
1st Monday, 12:30 P.M., Doctors' Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiolpulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs, 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical—Surgical Rdiological Conf.
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.
OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Genetics Conference
Weekly Monday, 12:30 P.M., Pediatric Department
Pediatric Rounds
Weekly Monday, Wed., & Friday, 10:30 A.M., Pediatric Department
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.
ECG. Conference
Weekly Tuesday, 12:30 P.M., Pediatric Department
Medical Grand Rounds
Weekly Wednesday, 8 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 8 A.M., PIMC
Visiting Professor Informal Rounds
Weekly Thursday, 9:30 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 12:30 P.M., PIMC.
Nephrology Conference
Weekly Friday, 12:30 P.M., Pediatric Department

ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N Wilmot Road, Tucson, AZ. Contact. Eric G. Ramsay, M.D. Approved for Category 1 credit.
Family Practice Department Meeting
3rd Tuesday, 12 P.M., Contact Wm. Moteforte
Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 P.M., Contact: Kim Sowards.
Current Concepts in Medicine
Weekly Tuesday, 12 P.M., Auditorium.
Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 P.M., Contact: S. Salmon, M.D.

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.
Cardiac Conference
Weekly Monday, 12:15 P.M., Auditorium
Surgery Conference
1st Tuesday, 12:15 P.M., Auditorium
Emergency Medicine Conference
1st Wednesday, 12:15 P.M., Auditorium
Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 P.M., Auditorium
Psychiatry Conference
3rd Thursday, 7 A.M., Auditorium
Combined Medical-General Practice Conference
1st Friday, 12:15 P.M., Auditorium
Toxicology Grand Rounds, Auditorium
1st Friday, 7:30 A.M., Auditorium
Ophthalmology Conference
1st Saturday, 8:30 A.M., Auditorium.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Emergency Medicine Lectures
Weekly Thursday, 8 A.M., Century Room A
Mental Health Update
1st Friday, 11:30-1:00 P.M., Century Room A
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Cardiology Conference
Weekly Monday, except last, 12 Noon, Contact: J. Fernadez, M.D.
Dermatology Conference
4th Monday, 5:00 P.M., Contact: R. Miller, M.D.

Endocrinology Conference
Last Monday, 12 P.M., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.
Chest Conference
3rd Tuesday, 12 Noon, Contact: William Nevin, M.D.
Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Infectious Disease Conference
2nd Tuesday, 12 Noon, Contact: B. Friedman, M.D.
Nephrology Conference
1st Tuesday, 12 Noon, Contact: Dr. Seltzer.
Orthopedic Conference
1st Tuesday, 8:00 P.M., Contact: Jay Katz, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 P.M., Contact: Dr. Lightner.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.
Clinical Pathology Conference
Last Wednesday, 8:00 A.M., Contact: Dr. Fuchs.
Family Practice Meeting
2nd Wednesday, 12:30 P.M., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.
Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buschsbaum, M.D.
Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.
Tumor Conference
Weekly Thursday, 12 Noon, Contact: Dr. Hastings.
G—I Conference
Weekly Friday, 12 Noon, Contact: C. Krone, M.D.
Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.
OBGYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.
OBGYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.

Surgical Department Confernece
Weekly Saturday, 7:30 A.M., Contact: Dr. Hastings, TMC.
Surgical Grand Rounds
Weekly Saturday, 9:00 A.M., UAHSC-Room 5403, Contact: Dr. Norton

U OF A HEALTH SCIENCES CENTER

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Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact: Drs. Otto & Zehngut.
Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 P.M., HSC Auditorium, Contact: Cancer Center
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Room 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.
Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn
Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.
Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson
Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.
Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford
Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room
Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross
Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.
Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley
Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.
Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.
Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital
Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.
Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.
Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson
Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655
Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich
Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.
Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology
Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.
Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.
Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.
Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.
Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.
Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.
Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.
Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.
General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

PHOENIX VETERANS ADMINISTRATION MEDICAL CENTER

7th Street and Indian School Road, Phoenix, AZ 85012. Contact: Alfred Heibrumn, M.D. Approved for Category 1 credit.
GI X-Ray Clinical Correlation Conf.
1st & 3rd Monday, 1 P.M., Rm. C435, Contact: Dr. Sanowski, Ext 618.
Medical/Surgical GI Conference
1st & 3rd Monday, 3 P.M., Rm. 3134, Contact: Dr. Kozarek, Ext 413. Dr. Mertz, Ext 493.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 P.M., RM. 3134, Contact: Dr. Mertz, Ext 493.
GI Pathology Conf.
2nd & 4th Monday, 1 P.M., Path Conf. Rm, Contact: Dr. Sakowski.
Vascular Conference
2nd Monday, 3 P.M., Room 3134, Dr. Cintora, Ext 419
Surgery/Pathology Conference & Tissue Review
1st & 3rd Tuesday, 4 P.M., Room 2128, Contact: Dr. Landard
Surgery Mortality/Morbidity Conference
4th Tuesday, 4 P.M., Room 3134, Contact: Dr. G. Mertz.
GI Grand Rounds
Weekly Tuesday, 1 P.M., Contact: Drs. Sanowski, Kozarek & Partyka
Cancer Symposium
Weekly Wed., 1 P.M., T-5, Contact: Dr. Byrne, Ext. 426.
Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 P.M., Contact: Dr. Rohwedder, Ext. 388.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 P.M., Rm 3134. Contact: Dr. Mertz, Ext. 493.
Medical Surgical Chest Conference
1st & 3rd Thursday, Contact: Dr. Rohwedder or Dr. Gill.
Cardiology Conference
2nd Thursday, 1 P.M., Rm. T-5.
Medical Service Grand Rounds
Weekly Friday, 11 A.M., Contact: Dr. Zeller
Surgical Journal Review & Videotape Session
Weekly 1 P.M., Room 3134.

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Medical Lectures
1st & 2nd Thursday & Weekly,Fridays, 1:30 P.M., Bldg. 28. Contact: J. W. Fitzharris, D.O.
GI Rounds
Third Thursday, 10 A.M.-2:30 P.M., Contact: J. W. Fitzharris, D.O.
Medical Rounds
First & seond Thursday, 1:30 P.M., Bldg. 28, Contact: J. W. Fitzharris, D.O.

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Weekly Tuesday, 2 P.M., Contact: Dr. Young
Medical Grand Rounds
Weekly Wed., 12-1:00 P.M., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam
Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.
Grand Rounds
Weekly, Thursday, 11 A.M., Bldg. 107, Contact J. Fitzharris, D.O.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon.
Neurology Grand Rounds
Weekly Friday, 12 P.M., Contact: Dr. Sibley.

YUMA REGIONAL MEDICAL CENTER (U of A, Tucson/ArMA)

2400 Avenue A., Yuma Az 85364. Contact:Alan Winfield, M.D. Approved for category 1 credit.

Radiology Conference
1st Tuesday, 7:00 A.M.
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2nd Tuesday, 6:30 P.M.
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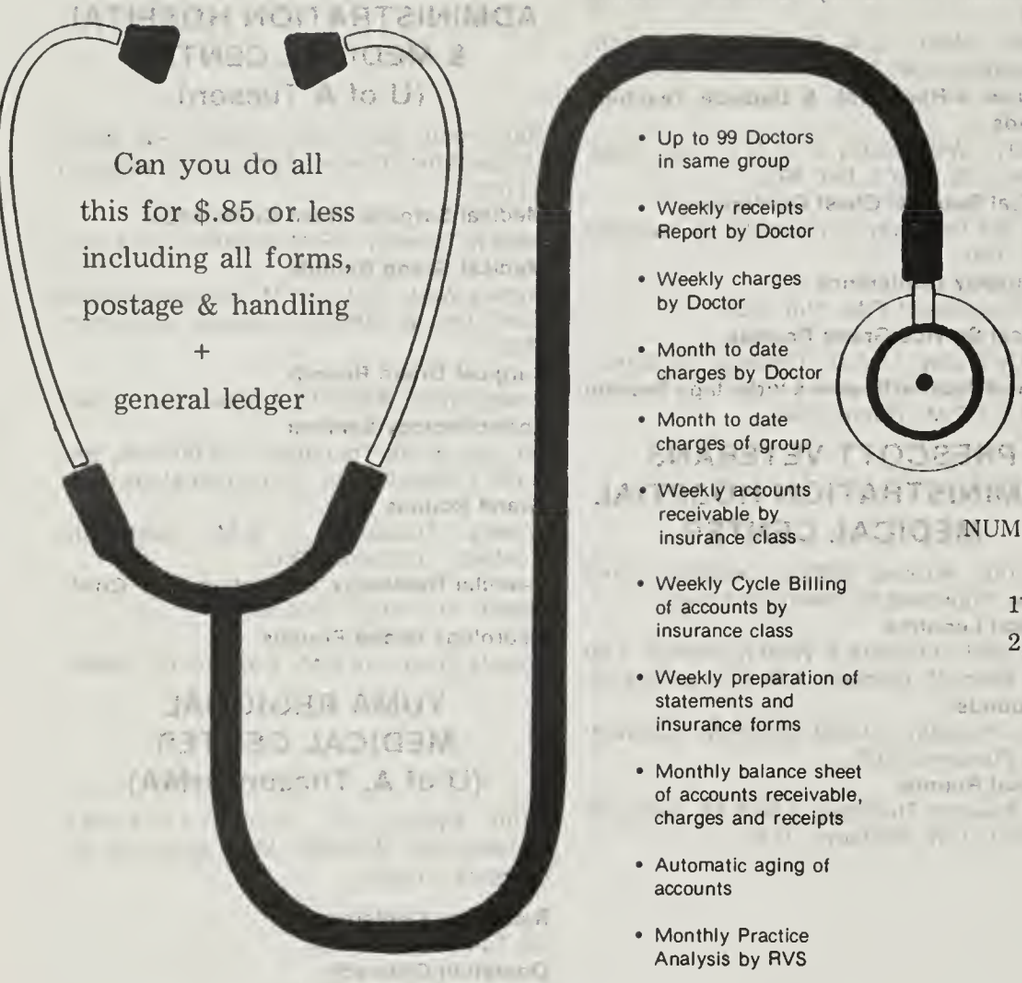
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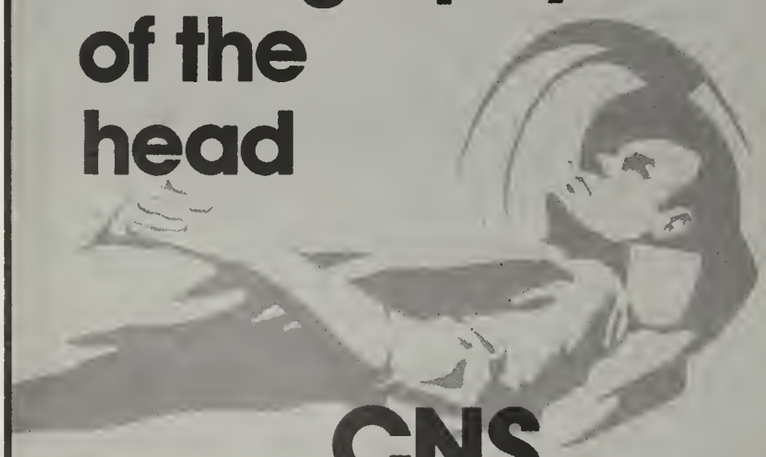
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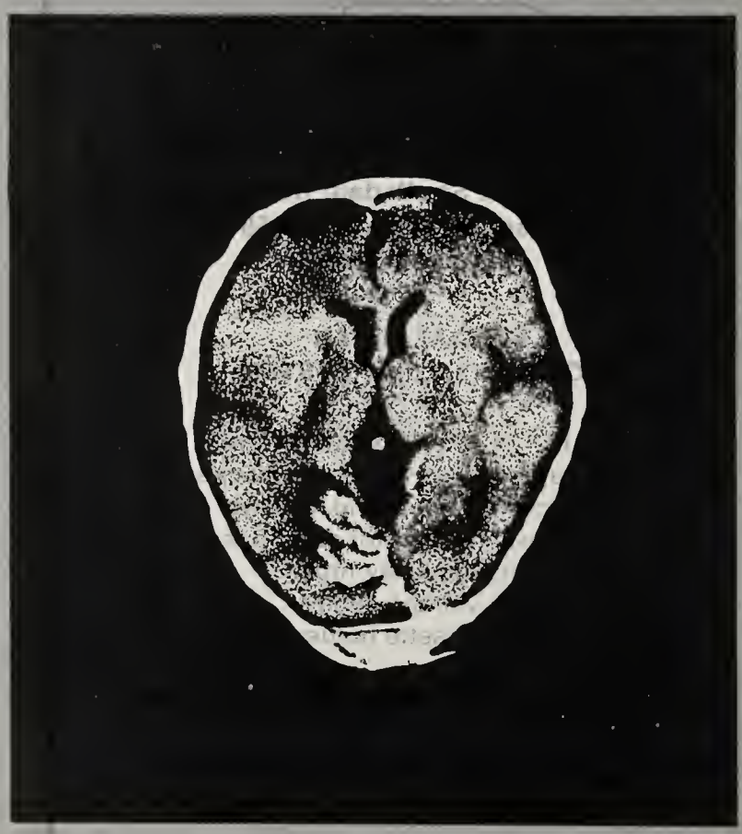
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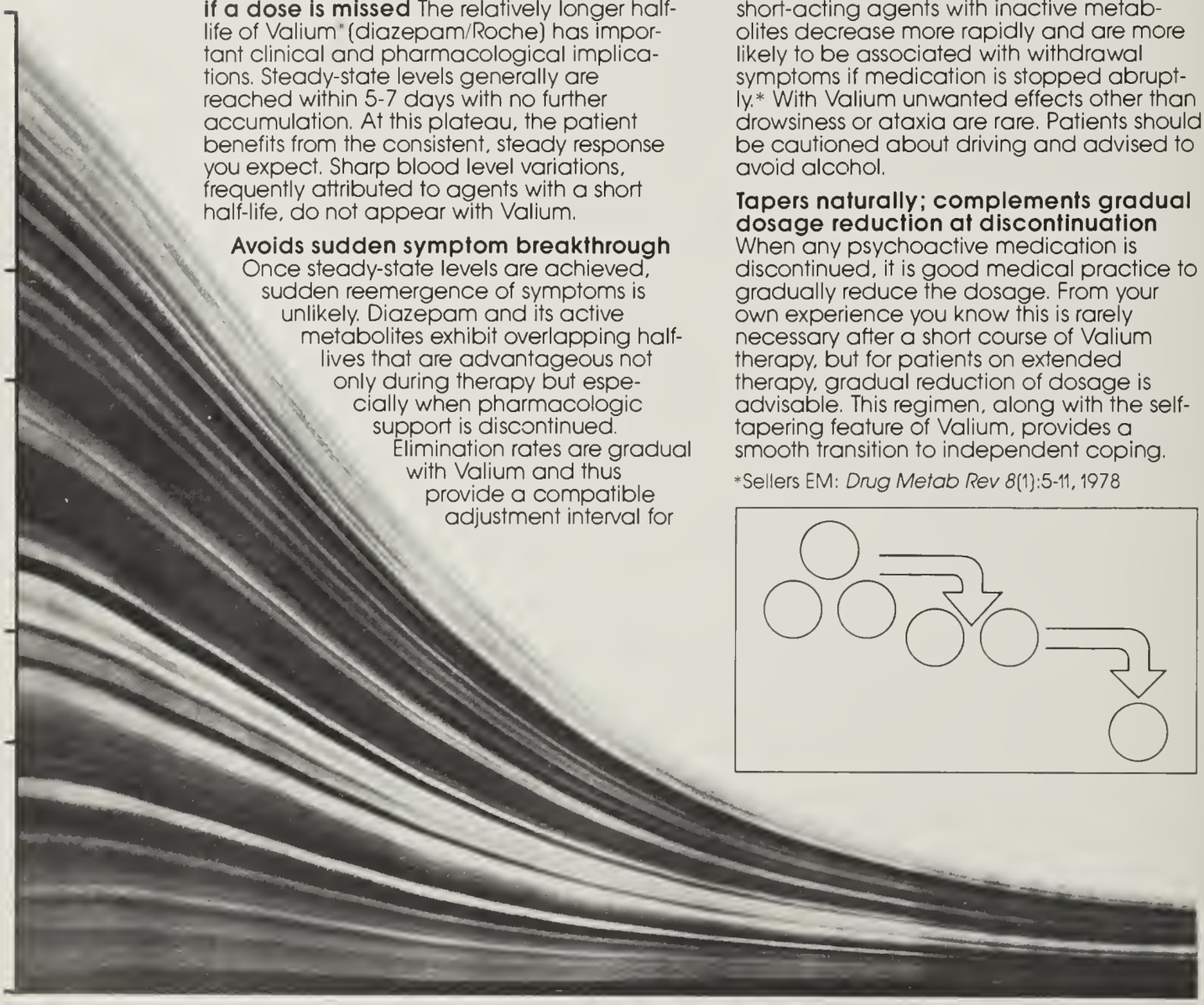
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.. XXXVII, NO. 9

half-life

Just one built-in advantage



Ensures smooth therapeutic effect even if a dose is missed The relatively longer half-life of Valium® (diazepam/Roche) has important clinical and pharmacological implications. Steady-state levels generally are reached within 5-7 days with no further accumulation. At this plateau, the patient benefits from the consistent, steady response you expect. Sharp blood level variations, frequently attributed to agents with a short half-life, do not appear with Valium.

Avoids sudden symptom breakthrough Once steady-state levels are achieved, sudden reemergence of symptoms is unlikely. Diazepam and its active metabolites exhibit overlapping half-lives that are advantageous not only during therapy but especially when pharmacologic support is discontinued. Elimination rates are gradual with Valium and thus provide a compatible adjustment interval for the patient. In comparison, blood levels of short-acting agents with inactive metabolites decrease more rapidly and are more likely to be associated with withdrawal symptoms if medication is stopped abruptly.* With Valium unwanted effects other than drowsiness or ataxia are rare. Patients should be cautioned about driving and advised to avoid alcohol.

Tapers naturally; complements gradual dosage reduction at discontinuation When any psychoactive medication is discontinued, it is good medical practice to gradually reduce the dosage. From your own experience you know this is rarely necessary after a short course of Valium therapy, but for patients on extended therapy, gradual reduction of dosage is advisable. This regimen, along with the self-tapering feature of Valium, provides a smooth transition to independent coping.

*Sellers EM: *Drug Metab Rev* 8(1):5-11, 1978

in the management of symptoms of anxiety

Valium®(IV)

diazepam/Roche

2-mg, 5-mg, 10-mg scored tablets

effective therapy through efficient pharmacodynamics

Before prescribing, please see summary of product information on next page

ROCHE®

Valium®IV
diazepam/Roche

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Management of anxiety disorders, or short-term relief of symptoms of anxiety, symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal, adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis, stiff-man syndrome, convulsive disorders (not for sole therapy)
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies The physician should periodically reassess the usefulness of the drug for the individual patient
Contraindicated: Known hypersensitivity to the drug Children under 6 months of age Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy
Warnings: Not of value in psychotic patients Caution against hazardous occupations requiring complete mental alertness When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication, abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures Advise against simultaneous ingestion of alcohol and other CNS depressants Withdrawal symptoms similar to those with barbiturates and alcohol have been observed with abrupt discontinuation, usually limited to extended use and excessive doses Infrequently, milder withdrawal symptoms have been reported following abrupt discontinuation of benzodiazepines after continuous use, generally at higher therapeutic levels, for at least several months After extended therapy gradually taper dosage Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence
***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed, drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action Usual precautions indicated in patients severely depressed or with latent depression, or with suicidal tendencies Observe usual precautions in impaired renal or hepatic function Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation
Side Effects: Drowsiness confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity insomnia, rage, sleep disturbances, stimulation have been reported, should these occur, discontinue drug Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy

ROCHE
Roche Laboratories
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Nutley, New Jersey 07110

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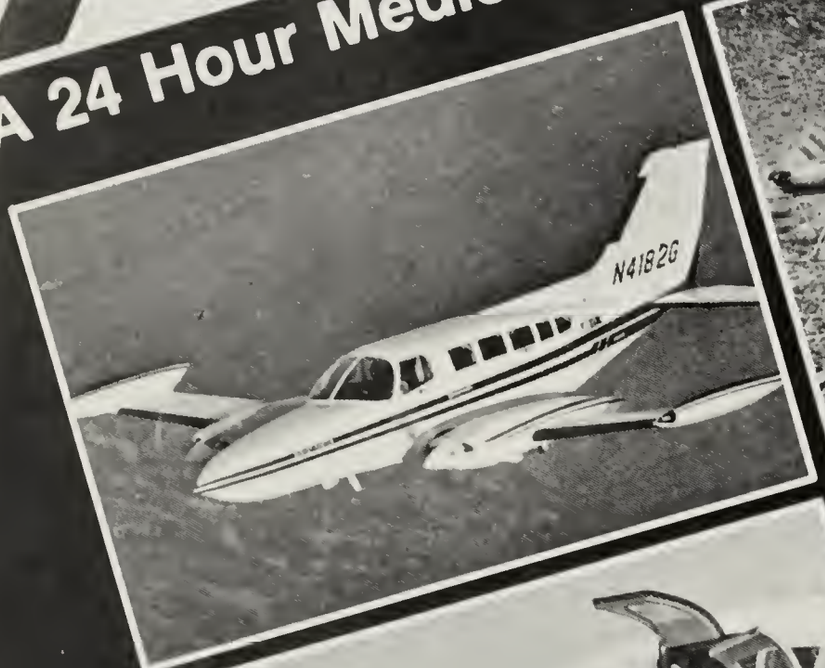
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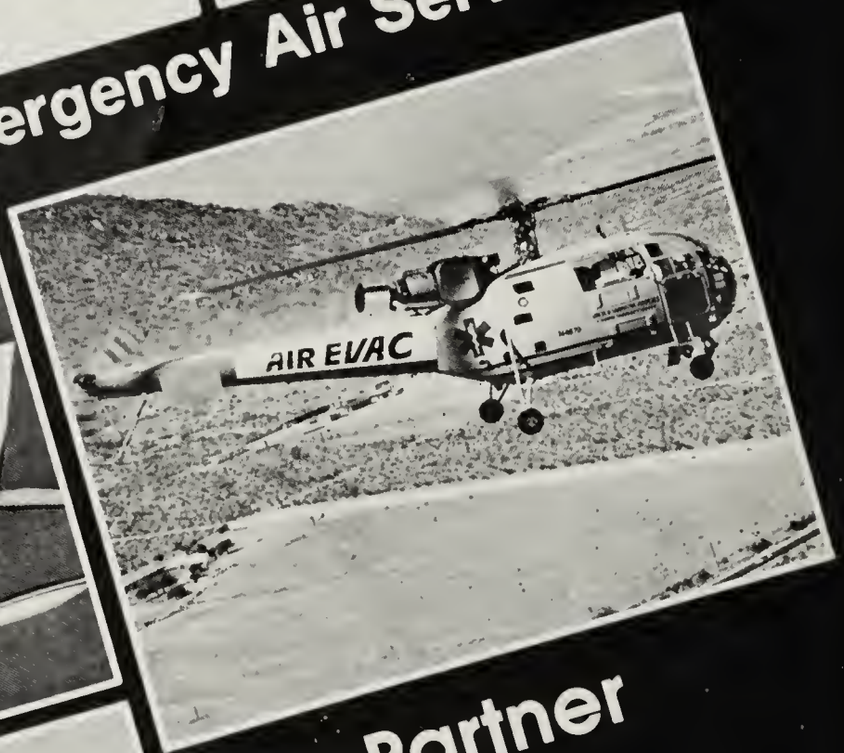
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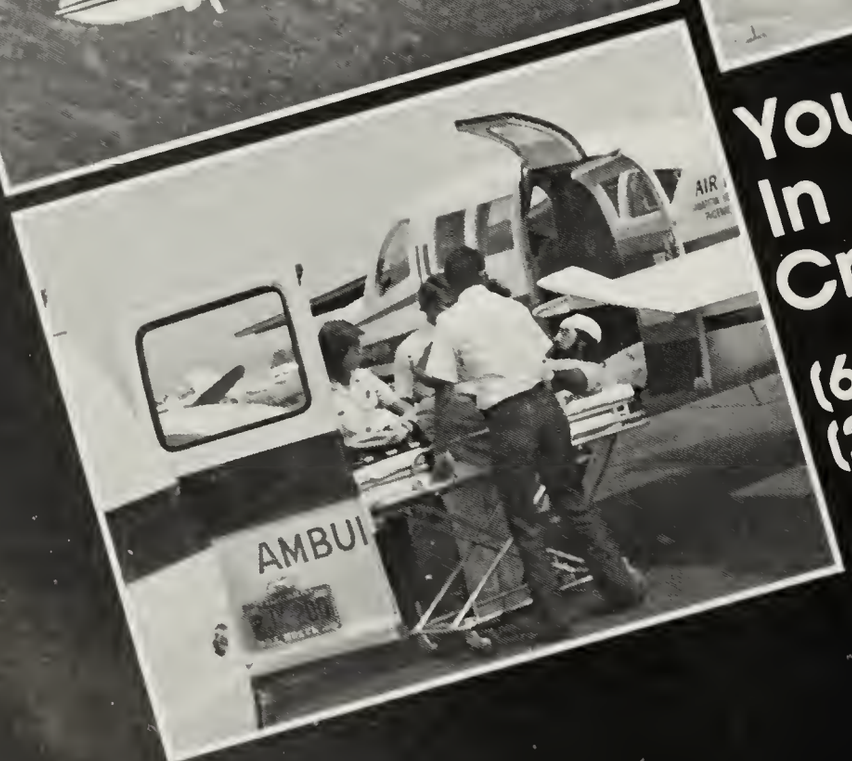
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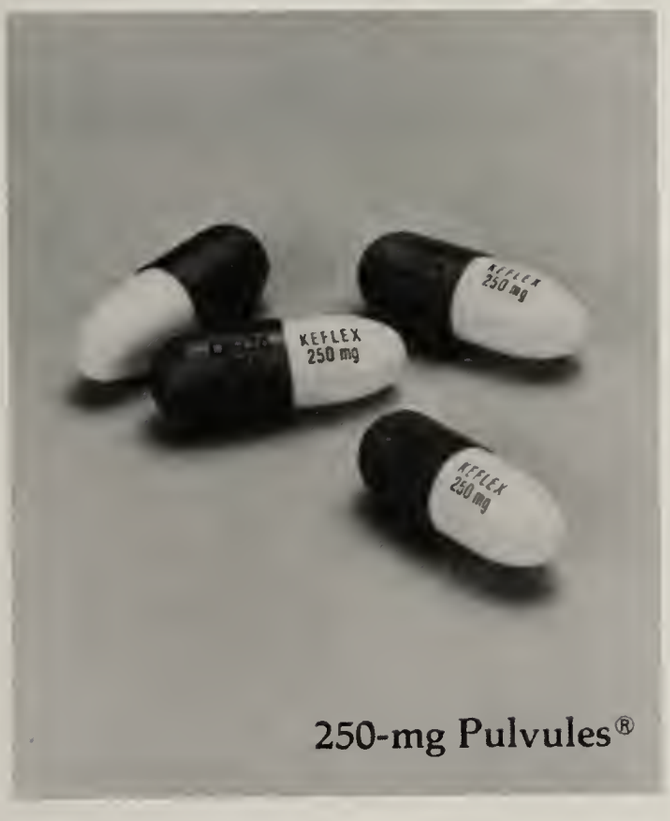
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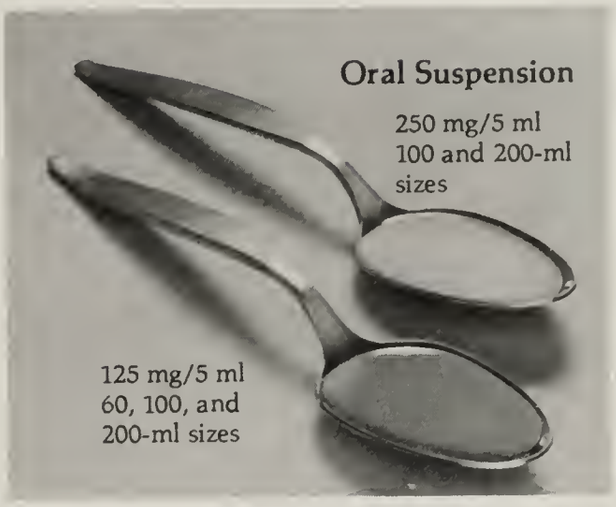
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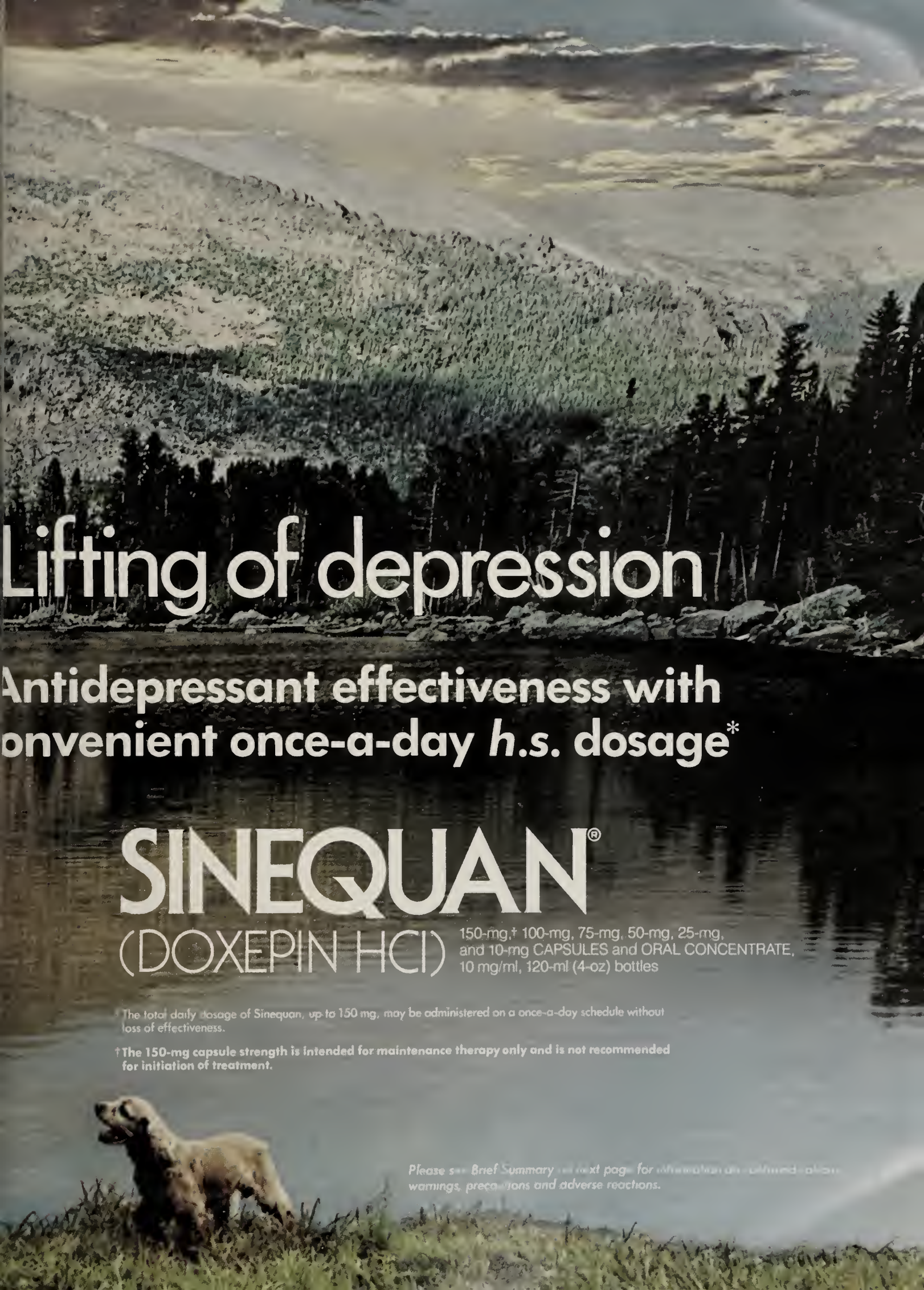
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(DOXEPIN HCl)
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*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.
†The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.
Please see Brief Summary on next page for
warnings, precautions and adverse reactions.

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150-mg,* 100-mg, 75-mg, 50-mg, 25-mg, and 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, 120-ml (4-oz) bottles

***The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Convenient once-a-day *h.s.* dosage

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule up to 300 mg per day.

Prominent sedative effect

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

Established antianxiety activity

to help alleviate the anxiety which often accompanies clinical depression.

Usually well tolerated

at doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

Extended range of dosage strengths

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BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate
Contraindications. SINEQUAN is contraindicated in individuals who have shown hypersensitivity to the drug. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

SINEQUAN is contraindicated in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies have been performed in rats, rabbits, monkeys and dogs and there was no evidence of harm to the animal fetus. The relevance to humans is not known. Since there is no experience in pregnant women who have received this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: The use of SINEQUAN in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with SINEQUAN. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered, and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of the possibility and cautioned against driving a car or operating dangerous machinery while taking the drug. Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects. Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular. Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic. Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic. Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.
A. Signs and Symptoms
1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment
1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade,® lemonade, orange juice, sugar water, Tang,® or water, but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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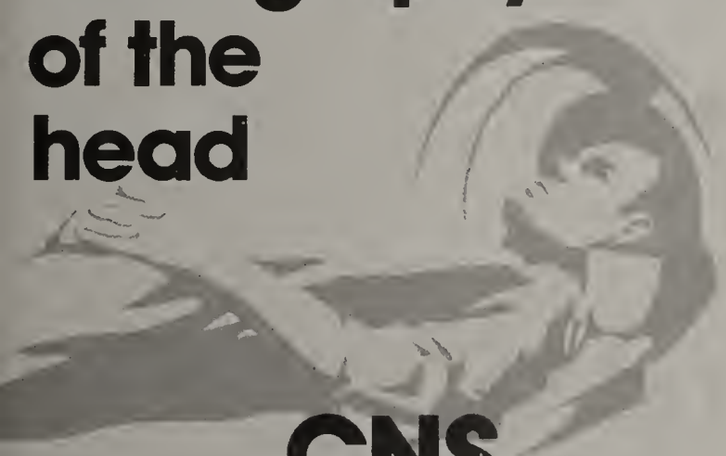


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Tenuate® (IV)
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly. *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico 00633

Direct Medical Inquiries to
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M. A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

Merrell

9 4672 (Y957A)

Overweight may not always be simple... complications can develop.*
Complicated or not...

Tenuate® Dospan® ℃IV
(diethylpropion hydrochloride NF)
75 mg. controlled-release tablets



A useful short-term adjunct in an indicated weight loss program.

Overweight patients in certain diagnostic categories often require strict appetite control and a successful program of weight reduction may tend to diminish the incidence or severity of the complications in some patients. Diethylpropion hydrochloride has been reported useful in such patients and while it is not suggested that Tenuate itself in any way reduces the complications of overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. **Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on the opposite page.**

In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness.

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate—it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

Merrell

For prescribing information see opposite page

In G.I. therapy



Adjunctive Librax®

Each capsule contains
5 mg chlordiazepoxide HCl
and 2.5 mg clidinium Br

antianxiety/antisecretory/antispasmodic

for adjunctive therapy of duodenal ulcer*
and irritable bowel syndrome*

Librax®

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

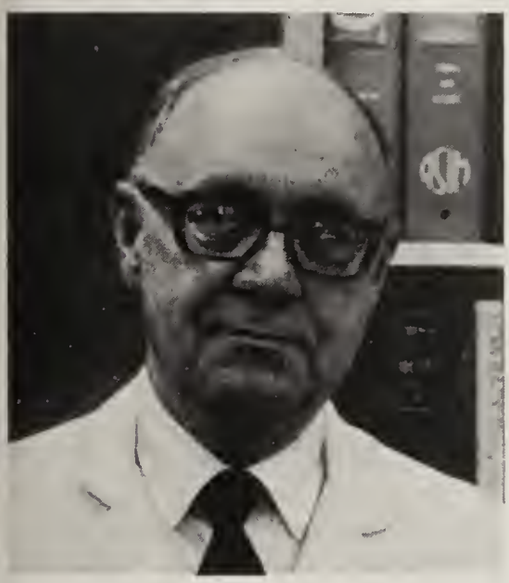
Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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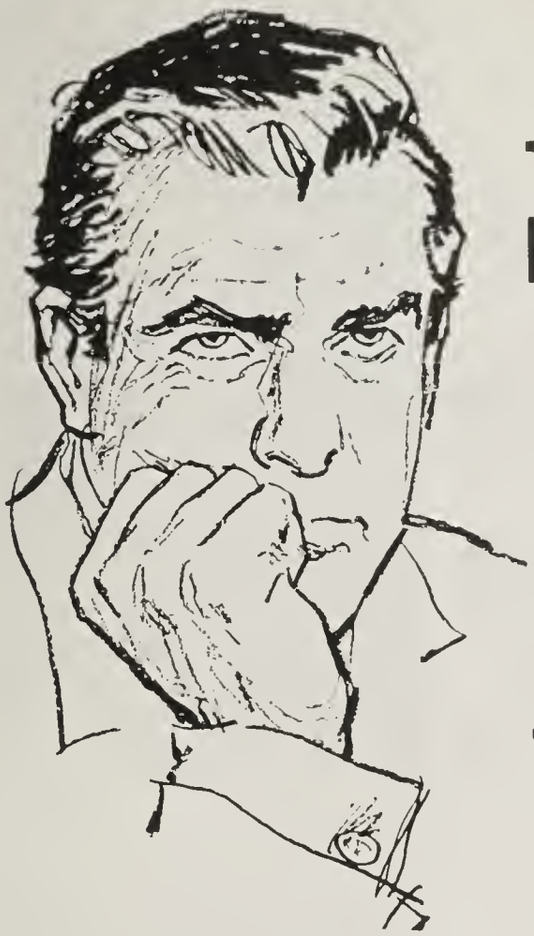
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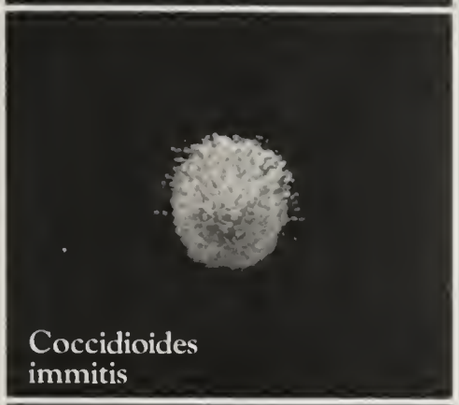
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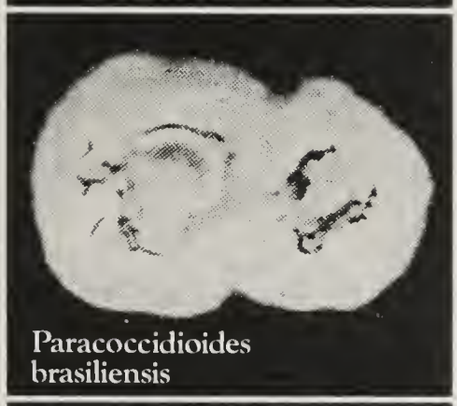
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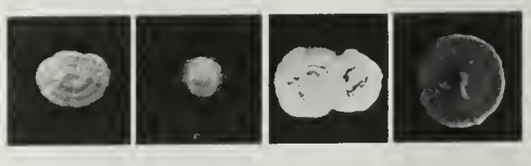
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CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v. is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in-vitro antifungal activity of MONISTAT i.v. is very broad. Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis, Candida albicans, Cryptococcus neoformans, and Paracoccidioides brasiliensis.*

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1µg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MIC's should be determined

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v. may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

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September 1980

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v. therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported. If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle, Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued.

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported. In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated

DOSAGE AND ADMINISTRATION:
DOSAGE
Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended:

Organism	Dosage Range *	Durotian of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to >20
Cryptococcosis:	1200 to 2400 mg per day	3 to >12
Candidiasis:	600 to 1800 mg per day	1 to >20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to >16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children. A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium.

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Hospitalization can be extremely helpful in breaking the binge eating-vomiting cycle that affect many anorexics. Camelback Hospitals offer a highly specialized approach to diet and nutrition, along with the services of a professional medical house staff ...on call 24 hours a day.

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- Acute psychiatric care for adults
- Geriatric Treatment Program
- Family, group and individual psychotherapy
- Occupational and recreational therapy — Psychodrama
- Social Services

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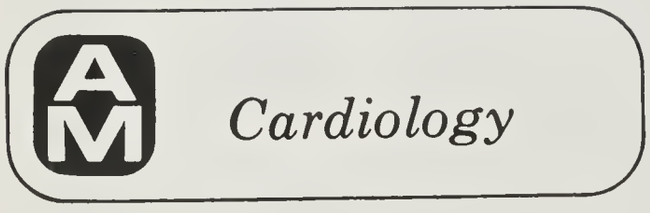
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Exercise Testing After Myocardial Infarction

Jay W. Smith, M.D.
Charles A. Dennis, M.D.
Frank I. Marcus, M.D.

Editor:
Kenneth B. Desser, M.D.

Most physicians when discharging a patient to home after a myocardial infarction will limit activity somewhat but, in general, will allow shaving, showering, driving and walking at two miles per hour. Therefore, it seems reasonable to exercise these patients in the hospital at a work load comparable to the activities of daily living anticipated immediately after discharge. If the patient does well in the controlled environment of the exercise testing laboratory he should do well at home. At least the test reassures the patient, the patient's family, and the physician that the patient is ready to be discharged and should do well at home.

In addition to its reassuring effect exercise testing may define cardiac risks induced by physicial activity-arrhythmias, angina, ischemic electrocardiographic responses-which can possibly be modified before hospital discharge. Although not yet proved, exercise testing may also have prognostic value.

At the University of Arizona the test is performed on an exercise treadmill the day before anticipated discharge. A modification of the Bruce protocol is used by exercising the patients for three minutes each at three different stages -zero degree grade at 1.7 mph, 10 degree grade at 1.7 mph and 12 degree grade at 2.5 mph. A V4-6 electrocardiogram (ECG) is continuously monitored and a 12-lead ECG is taken every 3 minutes, immediately after and 2, 4, 6, 8, and 10 minutes after exercise. The patient's blood pressure is recorded every three minutes during and after the test.

The test is stopped when the patient's heart rate reaches 60 percent of the predicted maximal heart rate calculated for age. Other reasons for stopping the test are falling blood pressure, angina pectoris, severe dyspnea, marked fatigue and ventricular tachycardia.

From: Sections of General Medicine and Cardiology, University of Arizona College of Medicine, Tucson, AZ 85724. Reprint requests to Dr. Smith, Department of General Medicine.

Most patients attain the desired heart rate, an average of 105 beats per minute. The test is not done in patients after myocardial infarction who have angina at rest, congestive heart failure, or neurologic or arthritic disorders that limit exercise performance.

The test can generally be considered safe. We have had no mortality in our series of over 135 patients and none has been reported in over 500 patients reported by others;[1] although, perhaps rarely, death can possibly be attributed to exercise testing in the patient recovering from myocardial infarction.[2] The total morbidity in over 600 patients tested was limited to ventricular tachycardia in seven patients,[1] and only one of these patients required cardioversion. Whether ventricular tachycardia induced by exercise should be considered a complication is debatable since one of the reasons for exercise testing is to identify high grade ventricular arrhythmias so that therapy can be initiated to prevent them.

Is exercise testing useful other than having a reassuring effect on the patient, the family and physician? In the first 62 patients tested at the University of Arizona, management was changed in 21 percent.[3] Therapy with antiarrhythmia medications was started or given in increased dosage because of arrhythmias developing during the test in four patients and an additional three developed angina requiring medications. Four patients remained in the hospital beyond the anticipated date of discharge as a result of ischemic changes or excessive responses of the heart rate to minimal exercise. Others were advised to severely limit their activity after hospitalization because of the results of the exercise test. Similar alterations in management were made by Erricson et al.[4]

Is exercise testing of any prognostic value in predicting sudden death or recurrent coronary events after hospital discharge? The following have been implicated as prognostic indicators during exercise testing: falling systolic blood pressure, ventricular premature beats (VPBs) and ischemic ST-segment responses.

The exercise stress test is an indicator of ventricular function in that a falling blood pressure during exercise reflects severe coronary disease,[5] and poor ventricular function.[6] Bruce et al[7] have reported that failure to attain a systolic blood pressure of 130mm Hg or greater with exercise was associated with a rate of sudden cardiac death greater than four times the rate in patients with maximum systolic pressure above 130 mm Hg. In our patients recurrent coronary events occurred much more commonly in those with falling systolic blood pressure during exercise.

Is the exercise stress test helpful in revealing VPBs and are these VPBs of prognostic significance? Clearly, VPBs measured by continuous recording late during the hospitalization[8] or after hospital discharge[9,10] have been related to poor prognosis. For instance, Vismara et al[8] reported that all patients who died suddenly had VPBs recorded in a Holter monitor before discharge from the hospital; there were no sudden deaths in patients without VPBs. Ruberman et al[9] reported sudden death in 15.5 percent of post infarction patients who had complex VPBs identified on a one-hour recording compared to 4.3 percent in patients without VPBs. However, the data on the incidence of coronary deaths following exercise induced VPBs are variable. Erricson et al[4] found a mortality of 10 percent at three months in patients with exercise induced VPBs and a mortality of 2.5 percent in patients with no VPBs during exercise. In addition, Granath et al[11] in following patients two to five years found a 24 percent mortality in 171 patients without VPBs compared to 47 percent mortality in 34 patients with VPBs. Markiewicz et al,[12] on the other hand, could not relate recurrent coronary events to exercise induced VPBs for recurrent events developed in four of 19 patients with VPBs and in three of 23 without VPBs in a follow-up period of 18 months. Our preliminary data reinforced other data showing that VPBs are important prognostically, especially VPBs immediately after exercise.

Exercise induced ST-segment depression has also been implicated as a prognostic indicator.[3] Markiewicz et al[12] found that 7 of 17 patients (41 percent) with ST-segment depression of at least 1 mm below baseline on at least one of the exercise tests done 3, 5, 7, 9 and 11 weeks following myocardial infarction had recurrent coronary events, while none of 25 patients without ST-segment depression had recurrent events. Theroux et al[13] reported a one year mortality of 2.1 percent in patients who had no ST-segment change compared to 27 percent mortality in patients with depression of the ST-segment. However, there was a false positive rate of 24.7 percent and most of the patients with ST-segment depression, 73 percent, did not die. Although reasonably sensitive, the test does not appear specific enough in identifying patients with a poor prognosis to justify high risk interventions.

In summary, exercise testing after myocardial infarction is safe and of psychologic benefit. It is useful in that clinical management is changed in approximately 20 percent of patients. Clearly, larger studies with long-term follow-up will have to be done before the clinical usefulness of these therapeutic interventions can be definitely known. The prognostic value of exercise testing following myocardial infarction is not known. In so far as it manifests poor left ventricular function and electrical instability it appears like a reasonable prognostic indicator.

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Editorial Comment
Kenneth B. Desser, M.D.

Performance of a modified treadmill exercise stress test prior to discharge of a patient with myocardial infarction taxes the courage of the practicing physician. Smith et al have presented compelling evidence that such a protocol may identify *selected* patients who are at risk for the development of potentially lethal cardiac arrhythmias. The presistence of resting S-T segmental elevation, depression or T wave inversion over the lateral percordial leads (V4-6) may nullify the significance of ST-T changes in these leads in those subjects who manifest abnormalities at the time of hospital discharge. A prospective study comparing a pre-discharge 24 hour ambulatory electrocardiogram with the exercise stress test in the same patient may shed light on the value of each procedure for predicting lethal coronary events. It may be that both in combination are worth the time, effort and expense. An inappropriate rise in diastolic blood pressure, albeit rarer, may have a prognostic significance which is the same as falling systolic blood pressure during the exercise setting. Exciting developments in therapeutic interventions which can prevent recurrent ventricular tachyarrhythmias are being published on a monthly basis. It is futhermore clear that a substantial number of patients with ischemic heart disease die as a result of ventricular arrhythmias which are unassociated with microscopic evidence of acute myocardial infarction on postmortem examination. Responsible physicians have an obligation to keep an open mind regarding any technic which may identify this population at risk. The work of Smith et al may provide one means of fulfilling this role.

Supported in part by the Biomedical Research Memorial Fund, Inc.

Chest Medicine

Difficulties in Weaning From Mechanical Ventilation
Positive End Expiratory Pressure and Supplemental Oxygen

Stuart F. Quan, M.D.
Faysal M. Hasan, M.D.

Editors:
Richard J. Lemen, M.D.
Bernard Levine, M.D.
John J. Seidenfeld, M.D.

A major therapeutic objective in the management of patients with acute respiratory failure is weaning from mechanical ventilatory support. The process of weaning is complex, and requires both an understanding of the physiologic abnormalities present in acute respiratory

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failure, and the changes which occur with weaning. When mechanical ventilation is withdrawn, tidal volume falls and respiratory rate increases; however, minute ventilation usually remains unchanged or decreases slightly.[1,2] Arterial oxygen tension (PaO_2) usually decreases, most frequently as a result of atelectasis and rarely from a diminished cardiac output.[1-4] A decrease in alveolar ventilation and an increase in the dead space to tidal volume ratio (V_D/V_T) during weaning leads to a rise in the arterial carbon dioxide tension ($PaCO_2$) and a decrease in arterial pH.[1,3,4] These changes are small and of little clinical significance in patients who wean successfully, but may be marked in those who are unable to wean.

It is the purpose of this paper to examine the causes of difficulty in weaning, review the clinical criteria useful in deciding when to initiate weaning, and to discuss the various methods of weaning from mechanical ventilation, positive end expiratory pressure (PEEP), and supplemental oxygen.

Common Difficulties In Weaning

Inability to sustain spontanous ventilation following mechanical ventilation is usually the result of one or more factors: progression of the underlying disease, hemodynamic instability, increased ventilatory requirement, decreased neuromuscular strengthen and excessive work of breathing. (Table 1)[5].

a) **Clinical Condition:** Progression of the patient's underlying disease (e.g. pulmonary edema, pneumonia, bronchospasm) is usually associated with weaning difficulty since weaning is unlikely to be successful when the clinical condition which resulted in respiratory failure is not improving. The patient should be alert, oriented and ready for weaning. Occasionally, patients may exhibit anxiety and fear related to the anticipated termination of mechancial ventilation. Ressurance and education, the use of intermittent mandatory ventilation *(vide infra)*, or biofeedback techniques may be helpful in these situations.

b) **Hemodynamic Status:** Weaning is usually very difficult in the presence of cardiovascular instability, and should probably be postponed until the patient's clinical condition is stable. Shock or hypotension and serious uncontrolled arrhythmias are absolute contraindications to weaning.[5] The simultaneous use of vasopressor agents is generally an indication of an unstable hemodynamic status, and in this situation the decision to wean should be made with caution.

c) **Increased Ventilatory Requirement:** The main etiology of an excessive ventilatory requirement is usually an increase in CO_2 production resulting from a hypermetabolic state related to fever, shivering, infection, and occasionally the need to provide respiratory compensation for a metabolic acidosis.[6] A large V_D/V_T ratio secondary to a primary process obliterating the pulmonary vasculature, or a low cardiac output may also increase the ventilatory requirement. In most patients, weaning should therefore be delayed until these factors are corrected. Occasionally, however, the use of vasopressors or aortic balloon counterpulsation may improve cardiac output and allow mechanical ventilation to be discontinued.[7]

Table 1
Common Difficulties in Weaning

Clinical Condition
Progression of Underlying Disease
Psychologic Adjustment
Hemodynamic Instability
Hypotension
Arrhythmias
Increased Ventilatory Requirement
Fever
Shivering
Infection
Metabolic Acidosis
High V_D/V_T
Decreased Neuromuscular Strength
Poor Nutrition
Electrolyte Disorders
Drugs
Excessive Work of Breathing
Low Compliance
Abdominal Distension
Bronchospasm
Excessive Bronchial Secretions
Respiratory Muscular Discoordination

d) **Decreased Neuromuscular Strength:** The major cause of inadequate neuromuscular strength is poor nutrition.[5,6] Nasogastric or parenteral nutrition should be started in patients soon after the onset of respiratory failure and institution of mechanical ventilation. In addition, many patients will benefit from a program of physical therapy to increase their muscle tone.[8] Such activities include passive and active range of motion exercises, and ambulation while being hand ventilated. Electrolyte disorders such as hypokalemia and hypophosphatemia, and the use of aminoglyoside antibiotics with muscle relaxant medications may also contribute to neuromuscular weakness and weaning difficulty.[5,6]

e) **Excessive Work of Breathing:** Increased work of breathing is a common cause of weaning failure. Generally, this is a result of a low respiratory system compliance caused by the underlying lung disease. Occasionally, there may be a reduction in compliance from extreme abdominal distension which may be relieved with nasogastric suction. Bronchospasm and excessive bronchial secretions may contribute to the high level of work required to breathe; they should be corrected prior to weaning. After prolonged periods of mechanical assistance, increased work of breathing may also be due to abnormal movement of the chest and abdomen resulting in asynchronous breathing.[6] This may be less of a clinical problem if intermittent mandatory ventilation is used.

Physiologic Criteria For Weaning

Initiation of weaning can usually be considered in patients who are clinically stable and show improvement in both their overall clinical condition and the disease process responsible for their acute respiratory failure. In addition, certain physiologic criteria should be met before weaning is contemplated. These can be divided into three general categories related to: a) lung mechanics, b) oxygenation, and c)ventilation.[6,9]

The most practical and commonly used tests of mechanical capability are the forced vital capacity (FVC) and the maximum inspiratory force (MIF). The FVC is an index of both neuromuscular strength and respiratory system compliance. For successful weaning, it is generally accepted that patients need to have a FVC of at least 10-15 ml/kg.[5,6,9,10] The MIF is an index to neuromuscular strength, and has the advantage of not requiring the cooperation of the patient. Most patients will not wean successfully unless the MIF exceeds 20 cm H_2O.[6] Other criteria which have been employed are a resting minute ventilation of 10 L/min,[11] the ability to voluntarily double the minute ventilation[11], a tidal volume exceeding 5 ml/kg[10], and a measured work of breathing less than 2.5 kg-m/min.[12] In most clinical situations it is possible to make a decision regarding the adequacy of ventilatory mechanics without the use of these other criteria.

The best measure of adequate oxygenation is the PaO_2. Patients should have a PaO_2 of 70 torr or more while breathing 40% oxygen or less, before weaning is contemplated. The use of 5 cms H_2O of positive end-expiratory pressure to maintain that PaO_2 is not a contraindication to weaning. In patients with chronic airway obstruction and hypercapnia, a lower PaO_2 is preferred to avoid suppression of the hypoxic ventilatory drive. Other reported criteria such as an alveolar-arterial gradient while breathing 100% oxygen ($AaDO_21.O$) less than 300-350 torr and an intrapulmonary shunt fraction ($\dot{Q}s/\dot{Q}T$ less than 10-20% generally offer no advantage over the measurement of the PaO_2. In fact, breathing 100% oxygen may itself increase $\dot{Q}s/\dot{Q}T$ and $AaDO_21.O$ by causing resorption atelectasis in areas of low ventilation to perfusion ratios.[13]

Adequacy of alveolar ventilation is clinically assessed by the $PaCO_2$ which should be maintained below 45 torr. Patients with chronic hypercapnea, should be ventilated at their "usual" $PaCO_2$ before starting weaning. Another criterion that may predict the outcome of weaning is the V_D/V_T ratio. Most patients with a V_D/V_T ratio exceeding O.6O are unable to sustain a level of alveolar ventilation adequate to prevent hypercarbia.[5,6] An exception, however, may be young individuals without a premorbid history of lung disease, who may wean successfully with a high V_D/V_T ratio.[6]

Methods of Weaning from Mechanical Ventilation

Two methods of weaning are presently employed:

a) The Conventional Method: Once it is determined that weaning criteria are fulfilled, sedation and enteral alimentation are discontinued early in the morning and spontaneous ventilation is initiated. Weaning usually follows a preconceived plan with progressively increasing periods of spontaneous ventilation interspersed with resting periods on the ventilator. During each weaning period, vital signs, arterial blood gases and physiologic parameters (FVC,MIF) are evaluated. Vital signs are recorded every 5-10 minutes for a half hour then every 30 minutes thereafter. FVC and MIF are measured hourly and at the end of the weaning period. If there is any evidence of respiratory distress or hemodynamic instability, arterial blood gases should be checked and mechanical ventilation resumed. Weaning should be attempted only during daytime with gradual lengthening of time off the ventilator. Later, weaning is extended through the night. Extubation can be performed after 4-24 hours of continuous spontaneous breathing if the patient is awake and alert.

b) Intermittent Mandatory Ventilation (IMV): IMV is a method of ventilation whereby a predetermined number of breaths are delivered by the ventilator while the patient is allowed to breath spontaneously between ventilator breaths.[14] As weaning efforts progress, the ventilatory rate is gradually reduced as long as the $PaCO_2$ is less than 45 torr and the pH is greater than 7.30, with little regard to other weaning criteria. One advantage of utilizing IMV is to maintain respiratory and abdominal muscular activity and thus possibly reduce the development of the abdominal-chest wall discoordination. Moreover, less observation by nursing and respiratory therapy is needed with IMV since the patient is not required to assume the entire work of breathing all at once. IMV may also be the preferred mode of weaning in patients who are psychologically dependent on the ventilator, since the portion of alveolar ventilation sustained by the patient can be progressively increased in a subtle fashion. In one study of patients weaning from short term mechanical ventilation following major surgery, weaning with IMV was more rapid than with conventional methods.[15] However, in patients who are difficult to wean, IMV has not been shown to offer any significant advantage. Some patients will wean only after combining periods of ventilation at low IMV rates with progressively lengthening periods of spontaneous ventilation.

Weaning From Supplement Oxygen and PEEP

In acute respiratory failure a high F_iO_2 may be needed to keep the P_aO_2 between 70 and 100 torr. The use of PEEP will allow the F_iO_2 to be reduced to avoid oxygen toxicity. It is not necessary to lower the F_iO_2 to less than 0.40, before weaning, since this is a level at which oxygen toxicity is not thought to occur. PEEP can then be safely reduced in 3-5 cms H_2O increments at 4-6 hour intervals when arterial oxygenation and respiratory system compliance show improvement.[16] However, premature lowering of PEEP may result in a significant

deterioration in P_aO_2 for greater than 24 hours, which may require a higher PEEP to correct than had been needed before the PEEP was lowered.[16] It is not necessary to lower PEEP to less than 5 cm H_2O before attempting to wean. Recent data indicate that use of PEEP will prevent the deterioration in P_aO_2 generally observed during weaning.[17] In fact, patients weaning from mechanical ventilation with PEEP may even experience an improvement in FVC and MIF.[17] Furthermore, patients can be successfully extubated while on low levels of PEEP without a deterioration in functional residual capacity or P_aO_2.[18]

Summary

Weaning from mechanical ventilation is one of the major goals in the treatment of acute respiratory failure. Although most patients can wean easily once their overall clinical condition improves, some patients will encounter difficulties in sustaining spontaneous ventilation. In these latter patients it is usually possible to identify the presence of one or more of several factors responsible for weaning difficulties. Correction of these factors and appropriate use of different weaning methods will allow these patients to wean successfully.

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Seminars in Gastroenterology and Liver Disease

Endoscopic Papillotomy for Common Bile Duct Stones

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Endoscopic papillotomy, a non-operative procedure for removal of common bile duct stones, is of particular value in the elderly and high-risk patient. Originally introduced for retained or newly-formed common duct stones in patients with previous cholecystectomy, indications have been expanded to include: common duct stones in high risk patients having an intact gallbladder, Sphincter of Oddi stenosis, and as a palliative maneuver in patients with biliary obstruction secondary to peri-ampullary neoplasm. Over the past year, we have undertaken this procedure in 20 patients with common bile duct stones and have achieved complete stone passage in 18 (90%). There was no associate mortality, and morbidity was limited to 1 episode of bleeding not requiring transfusion (5%) and 1 episode of clinical pancreatitis (5%).

Methods

Technically, endoscopic papillotomy should be undertaken only by an endoscopist with extensive experience in retrograde cholangiopancreatography. The procedure itself must be done in a fluoroscopy suite, and is accomplished using the side-viewing duodenoscope and patient sedation with meperidine or diazepam. The papillotome knife (Figure 1), consisting of a plastic cannula and an externalized cautery wire, is inserted freely into the common bile duct, and then pulled taut in the direction of the duct to form a cutting bow. A blend of cutting and coagulative current is thereafter passed along the wire and a 10-15 mm cut is made through the sphincteric mechanism of the distal common bile duct. If undertaken for common duct

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Figure 1
Side-viewing duodenoscope with bowed papillotome extended. Arrow delineates cutting wire.



Figure 2
Upper arrows demarcate 1.5 cm sphincterotomy incision, lower arrow points to the papillotome.

stones, these stones can either be allowed to pass spontaneously (2/3) or can be extracted with a Dormia basket or ballon catheter.

Case Report

A 67 year old female was admitted to hospital with a several week history of RUQ pain, recurrent fevers, and vomiting. Past medical history revealed a cholecystectomy for cholelithiasis 15 years prior, but was otherwise unremarkable. Physical examination revealed a toxic, elderly patient with a temperature of 40°C, pulse 120, and BP 100/62. Abdominal exam revealed a 12 cm tender liver and hypoactive bowel sounds. Laboratory data included a bilirubin of 1.4 mg/dl (nl<1 1.2), alkaline phosphatase 90 U/L (nl < 35), SGOT 120 U/L (nl <1 40), amylase 200 mg/dl (nl < 100), and a WBC count 21,000. Ultrasound of the pancreas and biliary tree was interpreted as within normal limits. ERCP delineated a normal pancreatic duct and dilated common and intraphepatic ducts with greater than 30 stones present in the ductal system. A 1.5 cm papillotomy incision was undertaken (Figure 2), and 15 stones retrieved from the common bile duct with a Dormia basket (Figure 3). The patient was subsequently restudied 4 days after papillotomy, revealing a widely patent sphincterotomy orifice (Figure 4) and three remaining stones in the common bile duct. These were readily extracted (Figure 5) with subsequent patient discharge the following day. She has remained asymptomatic in the 3 months post-discharge, and will be followed with an UGI in 6 months to assure patency of the sphincterotomy incision.

Discussion

Endoscopic papillotomy, developed simultaneously in Japan and Germany in 1973,[1,2] was first introduced into the United States by Geenen in 1976.[3] To date, over 5,000 patients in Europe and the United States have undergone this procedure[4] for indications which include; (1) retained or newly formed common bile duct stones post-cholecystectomy, (2) common duct stones in high risk patients with an intact gallbladder, (3) papillary stenosis, and (4) as a palliative procedure for periampullary neoplasms. An inability to ascertain the papillotome position, significant clotting abnormalities, long strictures of the common bile duct, and an uncooperative patient are all absolute contraindicatons to the procedure, while large stones (>2cm) and juxta-ampullary diverticulae are relative contraindications.

Success rate for endoscopic papillotomy ranges between 80-90% in most series.[4-8] Early complications including bleeding, pancreatitis, localized perforation, and stone impaction occur in 7-8% of individuals undergoing the procedure. Because 50% of those individuals developing a complication will require emergent surgery, endoscopic papillotomy must be coordinated with prior surgical back-up. Late complications of the procedure are few, although 1% of the patients done for common duct stones and 10-50% of papillary stenosis patients may restenose necessitating a repeat endoscopic or surgical procedure.[4,8,9]

Benefits of endoscopic papillotomy are most noteworthy in the high risk individual and the elderly. In the latter group, not only are common duct stones more prevelant, but they are also associated with a higher complication rate.[10,11] Moreover, because of concomittant cerebrovascular and cardiopulmonary disease, mortality for surgical sphincteoplasty or common duct exploration can average 4-8% in patients over 50.[13-15] In individuals such as these, endoscopic papillotomy offers a number of advantages: it can be done quickly (30-90 minute procedure time), requires significantly less hospitalization time, and its associated with little or no recuperative period. Because of these advantages, endoscopic papillotomy, where available, should be considered the treatment of choice in the elderly or high risk patient with amenable biliary tract disease.



Figure 3
Multiple stones are present in a dilated common bile duct. Arrows delineate endoscopic stone basket within the common duct.



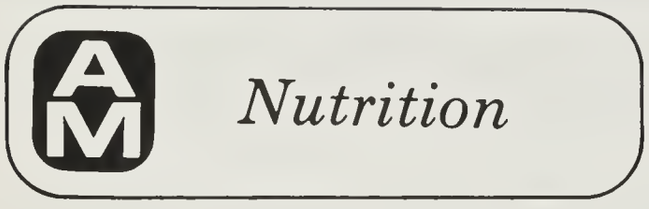
Figure 4
Widely patent sphincterotomy incision is outlined by arrows.



Figure 5
Common bile duct free of stones 4 days post-papillotomy. Contrast material is seen in the duodenum and some air is present in the biliary tree. Arrow represents incised sphincter segment.

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Management of Pancreatic Insufficiency

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Pancreatic insufficiency in the Western adult is most commonly the consequence of chronic pancreatitis due to alcohol abuse, although chronic pancreatitis may occasionally occur as a result of trauma or uncorrected biliary tract disease. Pancreatic resection for chronic pancreatitis or pancreatic cancer will also result in pancreatic insufficiency. The pancreatic insufficiency due to cystic fibrosis is the most common cause of malabsorption in childhood, and with the more prolonged survival due to modern treatment methods cystic fibrosis now constitutes a cause of malabsorption and malnutrition in adults. The general management of the chronic disorders which produce pancreatic insufficiency is beyond the scope of this article which will deal only with the management of the organ failure.

Patients presenting with a history and signs suggestive of malabsorption due to chronic pancreatitis with pancreatic insufficiency should have the malabsorption confirmed by a quantitative measurement of stool fat. D-xylose absorption will be normal, as will small bowel biopsy.[1] Vitamin B_{12} absorption may be depressed due to failure to digest the B_{12} binding R-protein.[2] The diagnosis may be confirmed by the findings of a calcified pancreas on a plain abdominal film, or by tests of pancreatic exocrine function.[3] The pancreas has a large functional reserve and mal-digestion does not usually occur until pancreatic exocrine secretion is reduced to about 10% of normal.[4] This explains the late appearance of fat malabsorption in patients with known repeated episodes of pancreatitis and with established pancreatic calcification. The malabsorption of chronic pancreatitis is largely of nutrients requiring luminal digestion, i.e., fat and protein. Bile salt secretion and intestinal mucosal integrity are normal and therefore the manifestations of malabsorption are less severe than those seen for example with celiac sprue. In particular, the bile salt dependent absorption of Vitamin D and other fat soluble vitamins is normal and bone disease is uncommon in pancreatic insufficiency.[5] Also, with the possible

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exception of Vitamin B_{12}, hemopoietic substance absorption is normal and anemia is not common.

About one-third of the patients with chronic pancreatic insufficiency have diabetes mellitus.[6] The diabetes is usually mild and easily controlled with insulin. Diabetic ketoacidosis, microangiopathy, and nephropathy are unusual, but may occur.

Management

The main principle in the treatment of chronic pancreatic exocrine insufficiency is to replace the deficient pancreatic digestive enzymes in order of allow the digestion and subsequent absorption of ingested food. This is achieved by the oral administration of pancreatic extracts, usually of porcine origin. The goals of such therapy are to prevent weight loss, or restore lost protoplasm to normal, to eliminate diarrhea, and in children, to establish adequate nutrition for growth. Achieving these goals has been more difficult than may be imagined and only in a minority of patients has normal fat assimilation been able to be achieved.[7] The problem is to get enough of the replacement pancreatic enzymes into the upper small intestine at the time food is passing through. Pancreatic enzymes, especially lipase, are susceptible to acid-peptic digestion and protection against such destructive hydrolysis in the stomach has been attempted by enteric-coating enzyme tablets or by administering antacids of Cimetidine with the enzyme preparations.[8]

Enzyme tablets or capsules must also be able to deliver the active enzyme at the appropriate time. For example, an enteric-coated tablet which carries its active ingredients with safety through the stomach but does not disintegrate until it has passed through the jejunum would do little to aid digestion. This may explain the finding of Graham that enteric-coated preparations were less effective than non-coated tablets or capsules in decreasing fecal fat excretion.[9] The lipase content of exogenous hormone preparations is the most critical in the control of symptomatic malabsorption. The majority of commercially available preparations contains insufficient lipase levels to be considered useful in pancreatic insufficiency. The non-enteric-coated preparations with the highest lipase content are: Ilozyme, Ku-zyme, Coatzyme, and Viokase.[9] Such preparations should be given as two tablets or capsules at the beginning of the meal, four single doses during the meal, and two at the completion of the meal.[10] There is no need to add bile salt supplements. A failure of such a regime to restore digestion to normal may be due to gastric digestion, and the addition of antacids, or Cimetidine may increase the therapeutic effect.

A new enzyme preparation is now available which has adequate enzyme levels packaged in enteric-coated granules (Pancrease, Johnson & Johnson). The recommended dosage of this preparation is two capsules with each meal. Initial impressions for this preparation show it to be a least as effective, if not more effective, than those products previously available and reflects the increased bio-availability of this preparation compared with earlier enteric-coated products. Its superior effectiveness, together with the more convenient dosage schedule makes it, in the writer's opinion, the best preparation currently available. The reduced number of capsules required, and the lack of need for simultaneously-taken antacids or Cimetidine to prevent peptic digestion of the enzymes, reduces its cost to below other available products and increases the likelihood of patient compliance.

Effective treatment of the pancreatic exocrine insufficiency with exogenous enzyme replacement allows more complete digestion and therefore absorption of undigested nutrients. This may exaggerate, or uncover previously undetected carbohydrate intolerance. Insulin dosages may therefore need to be adjusted upward to promote efficient assimilation of the increased nutrient load absorbed.

Some of the characteristics of chronic relapsing pancreatitis, i.e., pain, narcotic addiction, and pseudocyst formation are at least in part due to blockage of pancreatic ductular system. In selected instances such ductular obstruction may be overcome by surgical procedures such as the longitudinal pancreaticojejunostomy of Puestow, or the distal pancreaticojejunostomy of Duval.[11] It has been suggested that there may be some return of both exocrine and endocrine pancreatic function following these drainage procedures. However, data currently available are not sufficiently persuasive to recommend such surgical procedures as the primary treatment of pancreatic insufficiency.

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Oncology

Hairy Cell Leukemia

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In the two decades since hairy cell leukemia (HCL) was first described by Bouroncle et al,[1] its unique clinical, histologic, cytochemical, and therapeutic features have become clear. Because aggressive therapy appropriate for other lymphoproliferative disorders may be inappropriate or even detrimental to patients with HCL, awareness of its clinical aspects and diagnostic workup is crucial. This review will discuss hairy cell leukemia in the light of the many newly appreciated facets of the disease.

Clinical Findings

Most patients with HCL are men between the ages of 40 and 60 years. The male predominance is striking, with male-to-female ratios ranging from 3.7/1 to 7/1 in reported series[2-4] (mean age, 50 years; range, 22-86 years).

Presenting symptoms are non-specific, with easy fatigability being the most common (two thirds of cases). Bleeding, usually from the skin or mucous membranes, occurs in 20%-34% of patients. Recent infection was uncommon in the Utah series[3] but was found to occur in 38% of patients in the series of Golomb et al.[4] Abdominal fullness, skin disease, and central nervous system (CNS) complaints are other reported, but unusual, features. Of note is the uncommon occurrence of fevers, sweats, and weight loss, which are seen commonly with the malignant lymphomas.

Initial physical findings include splenomegaly in more than 90% of cases—often the only abnormal finding. Lymphadenopathy, on the other hand, is unusual, being present in about 20% of cases. Hepatomegaly is found in about 40% of cases, but never in the absence of splenomegaly. Purpura, pallor, and CNS signs are uncommon findings.

Routine blood count determinations reveal pancytopenia in about one half of cases, with anemia, thrombocytopenia, and leukopenia occurring individually in over 75% of cases each. Of importance, although leukocytosis does occur, it is unusual (15%). The blood smear commonly shows mild anisocytosis and poikilocytosis, consistent with a normochromic normocytic anemia. Findings of hemolytic anemia or a myelophthistic process are rare. The differential count will show neutropenia in most cases (78%) and frequently a striking lack of monocytes.[5] The characteristic "hairy cell" can be identified on careful examination of a Wright-stained peripheral blood smear in 90% of cases.[4] The "hairy cell" predominates in the 25% of patients in the leukemic phase of the disease. Other laboratory abnormalities include abnormal liver function tests in 10%-20%, elevated blood urea nitrogen (BUN) in 30%, elevated serum globulins in 18%, and abnormal protein electrophoresis in about 30% of patients. Only one patient with a monoclonal gammopathy has been reported.

Pathologic Findings

The "hairy cell" in a Wright-stained blood smear is distinctive. The cell is variable in size and may have scanty or abundant cytoplasm; however, the nuclear chromatin has a characteristically homogeneous or "ground-glass" appearance, with distinct nucleoli. This is easily distinguishable from the clumped chromatin of a normal lymphocyte. The cytoplasmic projections which give the cell its name are usually fine and delicate, but they may be thick. The projections are an unreliable guide to identification of the hairy cell, however, since they may be present as artifacts in other cells. Granules are unusual in hairy cells, although occasional azurophilic granules may be seen.

More conclusive identification of the hairy cell is possible with cytochemical techniques. Using electrophoretic techniques, Yam et al[6] found that the tartrate-resistant isoenzyme of acid phosphatase was present only in hairy cells; histochemical techniques have confirmed this specificity. Tartrate-resistant acid phosphatase (TRAP) has been rarely reported in the cells of chronic lymphocytic leukemia, lymphocytic lymphoma, Hodgkin's disease, and mononucleosis, but with much lower activity than that seen in hairy cell leukemia. Rarely, the TRAP test will be negative in patients who meet other criteria for HCL. Other cytochemical stains have been used, but they are not diagnostically helpful.

Bone marrow examination is always abnormal. Over 50% of attempts at bone marrow aspiration are "dry taps," but if a smear is obtained, it will show cells with the characteristic chromatin pattern of hairy cells. The cytoplasmic projections are often lost and may create a "dirty" background on the smear, due to cytoplasmic fragments. The core biopsy is usually hypercellular, with diffuse involvement of lymphoid cells. The cells

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appear less densely packed than those seen in the lymphomas. Reticulin is increased and a proportion of specimens show fibroblastic changes, with fusiform elongation of cells.[7] Mature collagen is not increased.

The nature of the hairy cell is not fully understood. It appears to have the immunologic and staining characteristics of a B lymphocyte, with some functional features (e.g., phagocytosis) of a monocyte. Electron microscopic observations are more consistent with a lymphocytic origin for the cell and often demonstrate a "ribosome-lamellar complex." This is a cylindrical structure with an open central area and multiple parallel surrounding membrane layers, with ribosomes lying between the membranes. The ribosome-lamellar complex may be a specific pathologic criterion for HCL; however, detailed correlative studies have not been carried out.

Differential Diagnosis

The absence of significant lymph node enlargement distinguishes HCL from other lymphoproliferative disorders, including malignant lymphomas, chronic lymphocytic leukemia, and Hodgkin's disease. In addition, the bone marrow biopsy specimen is less densely packed in HCL, and the cells stain for tartrate-resistant acid phosphatase in a characteristic manner.

Hairy cells may resemble the abnormal circulating lymphocytes seen in Sezary's syndrome, but the skin involvement associated with this syndrome is not seen in HCL.

Finally, the presentation of HCL may resemble that of chronic myelocytic leukemia, but morphologic and cytochemical assessment of the peripheral blood and bone marrow should allow an easy distinction.

Course, Complications, and Treatment

Hairy cell leukemia is a chronic disease in most cases. In the series of 71 cases reviewed by Golomb et al,[4] there was an overall survival of 51% at four years, with a levelling out of the survival curve thereafter. This suggests an excellent long-term outlook for about one-half of all patients.

Three principal manifestations determine the clinical course: 1) anemia, with attendant need for transfusion; 2) thrombocytopenia, with hemorrhagic problems; and 3) frequent infections. Anemia and thrombocytopenia may be due to both marrow replacement by tumor cells and hypersplenism. The degree of anemia or thrombocytopenia does not seem to bear a clear relationship to the prognosis of the disease.[4]

Infections are a common problem in HCL. Forty percent of the patients of Bouza et al[7] had serious infections, including septicemia and pneumonia due to *Pseudomonas* and *E. coli*. Fungal and protozoan infections occurred as well. Others have documented an increased incidence of tuberculosis in patients with HCL.

The etiology of this predisposition to infectious complications is multifactorial. Bouza et al[7] noted a clear association of infection with the degree of neutropenia and with prior steroid therapy. Patients with HCL also have poor marrow neutrophil reserve and leukocyte mobilization, perhaps because of circulating inactivators of chemotactic factors. Finally, some patients have defective lymphocyte blastogenic responses and antibody-dependent cell-mediated cytotoxicity; these defects may be correctable by leukocyte transfusion.

A variety of treatment regimens have been employed. About 20% of patients—usually those who are older and without severe cytopenias or frequent infections—will require no therapy. Corticosteroids alone are not helpful, and may be detrimental by increasing the risk of infection and shortening survival. Splenectomy improves thrombocytopenia and leukopenia in more than two thirds of patients[2,4] and probably reduces the risk of recurrent infections. However, in the largest reported series,[4] there was no clear survival benefit with splenectomy.

For patients who do not respond to splenectomy or who develop progressive disease thereafter (usually with marrow infiltration and failure), alkylating agents and aggressive chemotherapy regimens have been used with some recent successes.[8,9] Leukapheresis may be a temporary option for the patient with refractory disease.[10]

Conclusions

Hairy cell leukemia is a rare disease, with features that may suggest more common lymphoproliferative disorders. However, the distinction of this disease from lymphomas or other leukemias is not difficult on clinical and pathologic grounds.

Because most patients with hairy cell leukemia will do well with no therapy or with splenectomy alone, and may be adversely affected by aggressive programs, it is important that the diagnosis be suspected early and that appropriate workup be pursued promptly.

Acknowledgement

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Psychiatric Disorders

How to Talk to Your Patients About Sex

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Masters and Johnson indicate that one out of every ten married couples has a sexual problem, and if a person has a chronic illness, the chances are greater that he has a problem.[1] Interestingly enough, very few patients will discuss these problems with their physicians.

The physician can play an important role in helping his patients with their sexual concerns. He is frequently the only health care professional routinely seen by patients. Furthermore, he is seen as an authority figure who is knowledgeable and can be trusted. In a recent study, we found that seventy-two percent of women did not discuss their sexual concerns with their gynecologists.[2] Many of these women said they did not do so because of the physician's own discomfort with the topic. This is unfortunate because patients have no accurate and reliable source of information about sexuality.

Opening the Door to Discussion of Sexual Problems

How can the physician open the door to sexual discussion? His first task is to take stock of his own attitudes and feelings about sex in general. This will enable him to discuss sexuality in a nonjudgmental and realistic manner. This will be difficult to do, since like most people growing up in this culture, physicians have been taught not to talk openly about sex.

Just as it is difficult for the physician to bring up the subject of sex, it is also not easy for the patient. So the doctor should look for clues from his patients indicating possible sexual concerns. Physical complaints in the absence of disease, together with statements referring to declining physical abilities, may point to sexual problems. For example, patients may report "things are not going well at home" or "I guess my age is catching up with me." Patients may also allude to declining physical attractiveness, by making some derogatory comments about their looks or by appearing disheveled and withdrawn. Sometimes a patient may bring up a sexual problem as an afterthought, or as if it belongs to someone else, such as "By the way, I read that people who have my disease should not have sex" or "I have a friend who is unable to get it up any more."

The physician may weave the topic of sex within the context of a medical history. He can add questions about frequency of intercourse, erection and orgasm to his questions about bladder function. For women patients, questions about sexuality naturally follow when taking a menstrual history. In a relaxed and matter-of-fact way, he can say, "You know, you have never told me about your sexual functioning."

For hospitalized patients, the physician may also

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include the subject of sex in a list of others such as return to work, living arrangements, and so on. For example, he may say, "Now that you are going home, I expect that you will find your relationship with your wife somewhat changed" and ask more directly about sexual concerns. He may say, "You can resume all your routine activities now; go back to work, exercise, have intercourse, and so on."

Specific Counseling Skills

Once the physician has brought up the topic of sex, what can he do to encourage the patient to talk about his problems? The first step is to provide an environment where the patient can talk openly and freely about his sexual concerns. The physician can allow the patient to change into street clothes before discussing his specific concerns. To facilitate rapport and confidentiality, they can talk in the privacy of the office, where the doctor can give the impression that he has time and is concerned about the patient as an individual. The physician should not start if he does not have the time to thoroughly discuss a problem.

In discussing sexual concerns, you can make use of the 3 L's: *Listening, Language,* and *Labeling*. Listening involves both verbal and nonverbal behavior. You can indicate by eye contact and attentiveness that you are genuinely interested. Show that you are listening to the patient by not jumping in and asking questions, give the patient at least one minute to formulate his response before asking another question. Let the patient define the problem rather than having you define it for him.

Often patients find it difficult to discuss their sexual problems. You can help them "open up" by asking open-ended questions like "Can you tell me more about that? This type of question will elicit more response than a question like "Do you have a good sex life?", which will bring either a yes or no answer.

You can also word questions to imply acceptance of sexual behaviors, such as "Can you describe the erections you have when you masturbate?", "Is it only with your husband that you don't get turned on?" Be conscious of your sexual biases, thus reducing judgmental attitudes that may inhibit patients from confiding in you.

A Model for Office Management of Sexual Problems

In discussing sex, the use of technical language may intimidate or confuse the patients, as in this statement: "Does premature ejaculation inhibit your response during coital activities?" The physician needs to adopt an appropriate vocabulary level based on what the patient understands and what the doctor is comfortable saying, such as, "Do you come too fast during sexual intercourse?"

In addition, the use of certain medical terms serves to label a person. Clinical labels tend to create anxiety in the patient, making him feel that there is something abnormal about him. In particular, the labels "frigid" and "impotent" should be avoided. Instead, the physician can speak of difficulty in getting "turned on," aroused, or for men, having an erection.

A model for the treatment of sexual problems termed the PLISSIT model has been provided by Jack Annon in his book *Behavioral Treatment of Sexual Problems.*[3] It is a practical model that can be adapted to many clinical settings. The PLISSIT model of treatment has four levels of approach. Each letter or pair of letters stands for a suggested method of treating sexual problems. The four levels are Permission, Limited Information, Specific Suggestions and Intensive Therapy. Most patients can be treated by one of the first three levels of treatment, which can be viewed as *brief* therapy. The fourth level is *intensive* therapy. Each level of approach requires increasing degrees of knowledge, training and skill on the part of the clinician.

Level I: Permission Giving

The first level of treatment is *Permission Giving* which essentially gives the patient permission to have sexual thoughts, feelings and fantasies as well as to engage in sexual behavior. Sometimes people simply want to know from their physician that they are not "perverted" or "abnormal." For example, a doctor can reassure his patients that it is okay to masturbate and that many people do it. People frequently feel guilty for having erotic thoughts, fantasies or dreams about persons other than their spouses. The doctor can let them know that it is not unusual to fantasize during sex about someone other than their partner. Often, mothers feel ashamed of sexual feelings they experience when nursing their babies. Fathers frequently feel guilty about similar feelings when playing with their little girls. They can be reassured that these sexual feelings are a natural response to tactile stimulation. Older or disabled patients may also need permission to engage in sexual behavior.

Level II: Limited Information

Permission for sexual behavior is sufficient in a large number of cases to help the patient with his sexual concerns. However, in many cases, the physician may need to use the second level of treatment, *Limited Information*, which provides the patient with specific factual information related to his sexual concern. For example, many men worry that their penis is too small for the woman's sexual satisfaction. Letting them know that penis size is not important for producing orgasm and that most penises are about the same size in their erect state can relieve some anxiety.

One of the most prevalent misconceptions is the belief that the "normal" way for a woman to have an orgasm is through intercourse and that failure to do so reflects on her or her partner's adequacy. The doctor can tell the patient that the majority of women do not experience orgasm with intercourse alone because there is no direct clitoral stimulation. Many people need to be educated about the role of the clitoris in sexual arousal. A large number of individuals are not even aware of its existence. Providing this information can help countless couples engage in satisfactory sex and relieve feelings of inadequacy and frustration.

Level III: Specific Suggestions

Sometimes the knowledge gained from the limited information provided by the physician is not sufficient to resolve the patient's sexual concern. The physician can then either refer the patient elsewhere, or proceed to the third level of treatment: *Specific Suggestions*. This is a direct attempt to help the patient change his behavior in order to reach his stated goals. Specific suggestions can be given to overcome erection problems which have no organic basis. The doctor can suggest the use of the "stop-start" technique. He can instruct the patient to masturbate until he is almost ready to ejaculate, then stop and lose part of his erection. He is then to resume masturbating until he has another erection. Continued use of the "stop-start" method can provide the patient with the confidence that he can have an erection even after losing it. Once assured of his ability to maintain an erection, he can be more relaxed and confident with a partner. The "stop-start" method can also be practiced with a sexual partner or while fantasizing about one.

Another specific suggestion would be to engage in mutual sexual gratification with his partner up to the point where he experiences his *first* sign of anxiety. At that point he is to stop. They can relax, cuddle, have a glass of wine together and do whatever they would like; then start again.

For the patient with premature ejaculation, the "stop-start" method can also be suggested. The patient is to masturbate until he is conscious of the feelings prior to ejaculation, then stop and lose part of his erection. He is to resume masturbating until he again experiences the feelings prior to ejaculation; then stop. He can practice this several times a week until he learns to recognize and control the ejaculation. Specific suggestions for other sexual concerns appear in Annon's book.

Level IV: Intensive Therapy

In many cases, specific suggestions within a brief therapy format will alleviate most sexual problems. However, there will be times when the physician will need to refer the patient to intensive, long-term therapy. Patients should be referred when their problem has been unresponsive to treatment, or when it is related to a poor marital relationship.

Sexual functioning is an important component of mental and physical health. By providing permission, information and suggestions about sexuality, the physician can play a significant role in enhancing his patient's well-being.

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Case of the Month No. 45

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An elderly gentleman presented with a long history of atherosclerotic vascular disease. He had had previous aortic bypass surgery performed for this. Because of abdominal pain, enhanced computed tomography (CT) of the abdomen was performed. This demonstrated an unsuspected and asymptomatic urinary tract abnormality, later confirmed by excretory urography. His currently patent graft is also evident.

1) What is the urninary tract abnormality?
2) What type of vascular graft is present?

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Figure 1



Figure 2



Figure 3

Turn page for diagnosis →

Radiology Continued

Answer: 1) Retrocaval Ureter
2) Axillo-femoral Graft

Discussion:

Although computed tomography would not be used to assess this largely benign urinary tract anomaly, this case nicely defines the unsuspected finding. Figure 1 shows a CT scan after intravenous contrast at the level of the lower poles of the kidneys. The contrast filled right ureter (black arrow) can be seen passing posterior to the less strongly contrast-enhanced inferior vena cava. Note the axillo-femoral graft on the patient's right flank. A more inferior section (Figure 2) shows the ureter lying in the midline, medial to the inferior vena cava. The left ureter is normally positioned immediately anterior to the psoas muscle.

Retrocaval ureter was suspected in this patient by a urogram showing the abnormal configuration of the upper ureter. (Figure 3). Hydronephrosis and dilation of the upper third of the ureter may occur but not in this patient. Oblique projections usually show the ureter closely applied to the spine. Simultaneous inferior vena cavography and retrograde urography may be required to confirm the diagnosis. Retro-caval ureter is almost invariably a rightsided congenital anomaly. It was first described by Hochstetter in the 1890's, and Harrill is credited with the first radiographic diagnosis in 1940. Altered development of the inferior vena cava is responsible for the anomaly. Embryologically, venous return from the lower body drains via bilateral anterior and posterior cardinal veins. The infra-renal vena cava usually arises from the *right supracardinal* vein located dorsal to the ureter. The remaining venous channels normally obliterate. However, if the *ventral subcardinal* vein forms the definitive vena cava, a posterior or "retrocaval" right ureter results. Rarely, more than one channel persists producing double right or bilateral vena cavas.

Clinically, retrocaval ureter may present with symptoms of obstruction, with episodic right flank pain, occasional hematuria, and an increased incidence of calculi and infections. Many cases are diagnosed incidentally and repair is undertaken only if significant obstruction is identified.

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Renal Disease

Chronic Renal Failure Management — Part I

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Chronic Renal Failure (CRF) is characterized by slowly progressive deterioration of renal function over months to years, caused by a variety of diseases. These disorders may be exclusively renal, as in proliferative glomerulonephritis, a feature of a systemic or generalized metabolic disorder (Fabry's disease, diabetes) or obstructive urologic disease. The spectrum of CRF embraces a near normal functional state in an asymptomatic individual to profound uremic syndrome necessitating dialysis or transplant for survival.

This discussion is concerned with management of patients prior to the development of end stage renal disease (ESRD) i.e., those with a glomerular filtration rate (GFR) of 5-10 ml per minute or less and requiring dialysis—transplant intervention. Recognition and proper management of CRF in earlier stages is important and often critical in minimizing later complications, such as hypertensive vascular disease, and renal osteodystrophy.

The course of CRF is occasionally punctuated by abrupt decreases in renal function. Causes include sodium and water depletion, urinary tract infection, nephrotoxic agents, urinary tract obstruction, and hypotension due to excessive antihypertensive therapy. These causes are generally easily recognized and restoration of pre-existing renal function levels is generally the rule, with correction of the above conditions. Those patients with advanced CRF (GFR 10-20 ml per min) obviously cannot tolerate abrupt decreases in renal function without clinical deterioration and particularly require careful, prompt management, which may include interim dialysis support.

Nutrition-Metabolic Care

Nutritional and metabolic care is important. Adequate caloric intake, restriction of low biologic value protein, vitamin supplementation all play roles. Tissue weight loss is common due to anorexia, unduly restricted diets, and urinary protein losses. Use of highly restricted protein diet (Giovannetti-Giordano diet furnishing .3 gm per kg body weight or approximately 20 gm of high biologic value protein) has limited usefulness because of poor palatability and potential malnutrition with negative nitrogen balance with long term use. Protein restriction of .5 to 1.0 gm per kg body weight allows greater dietary flexibility, patient acceptance, and positive protein balance, with tissue weight maintenance and maintenance or rise in serum albumin. The degree of azotemia, metabolic acidosis and hyperphosphatemia, as well as clinical course, determine timing of the use of dietary modification. In general, patients with GFR of 25 ml per minute or more require little or no diet modification, except for sodium restriction particularly in the presence of hypertension or cardiac failure.

Increased use of carbohydrates and fats to offset protein restriction is often necessary to provide adequate caloric intake. Dietary supplements utilizing high carbohydrates and containing 1-1.5 cal per ounce, such as Precision Isotonic and Ensure, are useful supplements.

The carbohydrate intolerance seen in uremia rarely requires tratement except where massive glucose infusion is given, e.g. peritoneal dialysis or parenteral hyperalimentation. Insulin is the agent of choice for treatment of diabetes; sulfonylurea agents are contraindicated, especially long-acting drugs such as chlorporopramide (Diabinese) because of prolonged half-life and hypoglycemia.

Fasting hyperlipidemia with hypertriglyceridemia, type IV hyperlipoproteinemia, is seen frequently and characterized by defective lipoprotein lipase clearing in the face of elevated triglyceride production rate and a decreaed HDL fraction. These lipid abnormalities are of interest because of the accelerated atherosclerosis and coronary artery disease noted with CRF and ESRD. Treatment approaches include the use of carbohydrates and saturated fat restriction and drug therapy such as clofibrate. None of these approaches is entirely satisfactory; carbohydrate restriction further restricts

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caloric intake. Use of clofibrate requires dose reduction (500 mg per day) to avoid severe myopathy and other complications.

Water soluble vitamin supplements are useful where dietary restriction has been imposed. The B complex vitamins, folic acid, and vitamin C should be given. Use of vitamin D is discussed below.

Calcium, Phosphate, Vitamin D, and Metabolic Bone Disease

Disorders of calcium, phosphate and vitamin D metabolism result in a variety of metabolic bone disorders and alteration in calcium-phosphate blood levels. Defective active vitamin D synthesis, defective calcium intestinal absorption, phosphate retention due to decreased GFR, secondary hyperparathyroidism are major abnormalities. These result in osteomalacia-rickets, osteoporosis, osteitis fibrosa, and occasionally osteosclerosis. Bone pain and pathologic fractures are seen in more advanced situations. Chemically, hyperphosphatemia occurs at a GFR of 25 ml per minute or less, with hypo- or low-normal-calcemia the rule. Hypercalcemia is extremely rare, and should prompt search for other factors such as underlying malignancy, prolonged immobilization or unrelated disorders. Parathormone blood levels are uniformly elevated.

The goal of therapy is to promote calcium absorption, control hyperphosphatemia, to promote the healing of an osteomalacic skeleton, and to diminish and suppress parathormone release.

The following measures are indicated:

A. Control of hyperphosphatemia by limiting dietary phosphate, e.g. milk, and the use of phosphate-binding antacids such as aluminum hydroxide taken with meals.

B. Adequate calcium intake (1 gm per day in adults) per diet and calcium supplementation: calcium carbonate is especially useful where systemic acidosis and hypocalcemia both require treatment.

C. Vitamin D supplementation: Calcitrio (1, 25 dihydroxy vitamin D3) is the most biologically effective vitamin D analog currently available. Its use requires the frequent measurement of serum calcium and phosphate levels to avoid hypercalcemia, increased calcium phosphate product, undesired metastatic calcification.

Cardiovascular Complications

Major cardiovascular complications in CRF include coronary artery disease, hypertensive vascular disease, and uremic pericarditis. Accelerated atherosclerosis with premature coronary disease and peripheral vascular calcification are probably due to a number of factors including hypertension, hyperlipidemia, and metastatic calcium deposition in the vascular tree due to elevated calcium-phosphate product. The use of clofibrate and low carbohydrate diet in controlling hyperlipidemia has been mentioned above. Metastatic arterial calcification (rarely myocardial and cardiac conduction system calcification), may be minimized by control of hyperphosphatemia with phosphate-binding antacids; control of hypertension is probably the most important factor and is detailed below.

Increasing experience has shown that individuals with CRF can undergo coronary bypass surgery acceptably, but require special attention to electrolyte, fluid, and metabolic disorders. Patient groups that have especially high risk for severe coronary atherosclerosis are those with CRF due to diabetes mellitus.

Hypertension is a frequent complication of CRF. Mainstays of treatment are sodium restriction, anti-hypertensive drug therapy, and rarely, bilateral nephrectomy. Increased total body sodium and water, hyperreninemia and increased peripheral vascular resistance are pathogenetic factors. Individuals with chronic glomerulonephritis, polycystic kidney disease, diabetes mellitus, collagen vascular disorders, are especially likely to have hypertension.

Dietary sodium restriction is the initial step in treatment. If restriction is excessive, however, volume depletion, hypotension, and further fall in GFR may occur. Drug therapy can be instituted using "step therapy." Thiazides can be used effectively in mild CRF as a "Step I" drug. "Step II" drugs such as beta-adrenergic blocking agents (propanolol or metroprolol, methyl-dopa or clonidine) are useful adjuncts where thiazides alone are not effective. Furosemide or thiazides often are necessary to prevent fluid retention observed with step II drugs including methyl-dopa, clonidine and beta-blocking agents. "Step III" drugs include the vasodiolators (apresoline, prazosin, minoxidil) and guanethidine. The vasodilator group may be especially useful in reducing systemic vascular resistance in patients with low cardiac output states (reduction of afterload). In severe hypertension with advanced CRF, combination therapy using 1) a beta blocking agent, 2) minoxidil, and 3) diuretics, (furosemide or metazolone) to obviate fluid retention can be used. This is often an effective regimen and can reduce the need for bilateral nephrectomy. The latter procedure should be reserved for those patients who are A) severely hyperreninemic, B) the unresponsive or intolerant to aggressive drug therapy and C) have ESRD with contemplated renal transplantation.

Uremic pericarditis is a late complication to CRF. Widespread use of echocardiography has demonstrated the frequent presence of small to moderate pericardial effusion, frequently posterior, in asymptomatic azotemic patients. This finding is usually of little or no clinical importance.

Acute uremic pericarditis is usually painful, associated with left pleural effusions, and may progress to life-threatening cardiac tamponade. Therapy with indomethacin, corticosteroids (systemic or intra-pericardial), and institution of dialysis therapy are of benefit. Unresponsiveness to medical therapy cardiac tamponade, and progression to chronic constrictive pericarditis are indications for surgery. Radical pericardiectomy, in our experience, is the procedure of choice.

State of the Art

Snake Venom Poisoning:
Diagnosis and Treatment

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In most areas of the United States recognition of venomous snakes is difficult. Fortunately, in Arizona there are only two groups of poisonous sankes: the rattlesnakes *(Crotalus)* and the Arizona coral snake *(Micruroides euryxanthus)*. Bites by the Arizona coral snake are rare owing to its secretive habits and diminutive size.[1] There has been no recorded fatality resulting from the bite of the Arizona coral snake. Nearly all of the snake envenomations occurring in Arizona are caused by one of the rattlesnake species. It has been estimated that between 50-100 rattlesnake bites occur each year in Arizona. Fatal outcomes are uncommon.[2]

Diagnosis

Snake venom poisoning is a medical emergency that requires immediate attention and the exercise of considerable judgment. For years medical practices have been dictated by tradition, anecdotes, fear and ignorance. Several facts need emphasis. There are many nonpoisonous snakes that bite and these wounds require little, if any, therapy. Persons who have been bitten by any type of snake are usually apprehensive, and can be hysterical. Neither the patient nor the physician may be capable of exact species identification. The physician must rely on certain signs and symptoms when deciding whether a poisonous snake did the biting, and if so, whether venom was injected.

The bite site should be inspected for the presence of one or two fang punctures in addition to the rows of pinprick-size tooth marks common to poisonous and non-poisonous snakes alike. If no fang punctures are seen it can be assumed that rattlesnake envenomation has not occurred. Once it has been determined that a rattlesnake made the bite the physician must make an assessment of whether or not envenomation occurred. Between 20-40% of documented rattlesnake bites result in no venom being injected.[2-4] Finding fang puncture sites is not sufficient evidence to begin therapy.

Rattlesnake venoms differ in their composition between species. Numerous enzymes (hyaluronidases, proteases, phospholipase A, phosphodiesterase, esterases) are contained in rattlesnake venom and are responsible for the intense local inflammation and tissue

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damage. In addition, many complex polypeptide toxins mediate the neurotoxic, hemotoxic, and cardiotoxic effects of rattlesnake venom. The early signs that envenomation has occurred are edema spreading from the bite, local pain, local ecchymoses and perioral tingling or numbness. In moderate or severe poisonings, the edema progresses rapidly and may be accompanied by lymphangitis and painful regional lymph nodes. Ecchymosis is a constant finding and sero-sanguinous bleb formation is common. Systemic poisoning may be immediate or delayed several hours. The unpredictability of systemic illness, regardless of the serverity of local damage, makes hospitalization preferable for all cases of snake venom poisoning. Evidence for systemic envenomation includes nausea and vomiting, thirst, sweating, fever, shock, diffuse intravascular coagulation, hemorrhage from any site, paresthesias, respiratory paralysis, coma and seizures.[5]

Therapy

If the patient arrives within an hour after the bite, incision and suction of the fang puncture site is the local treatment of choice. In one study, up to 35% of experimentally injected venom was retrieved if incision and suction was begun within several minutes, and some retrieval was shown after a delay of 30 minutes.[6] McCollough and Gennaro,[7] using rattlesnake venom tagged with radioactive iodine, reported more than 50% recovery if incision and suction was started within three minutes of subcutaneous injection and stated that this procedure may be valuable as long as two hours after a bite. Each incision need only be linear, not cruciate, and no more than 1/2 inch in length and 1/8 to 1/4 inch in depth. This will minimize any additional tissue injury resulting from the incision. The incision or incisions should be made directly over each fang puncture site and if possible, should be made in the direction of fang travel. Suction can be accomplished with a breast pump or a syringe without the plunger, inverted, and attached to a suction outlet. In the field or in the absence of suction devices, suction can be safely performed by mouth. It should be continued for about 30 minutes. If incision and suction are begun promptly there is no apparent increase in the amount of retrievable venom when a tourniquet is applied proximal to the bite,[6] although Russell favors tourniquet use.[5] Vascular occlusion concentrates the many toxic fractions of the venom in the extremity and theoretically could increase local tissue damage.

On admission to the hospital, and in the presence of obvious findings of envenomation, the patient should be skin tested with horse serum (0.02 cc of a l:10 solution) in anticipation of treatment with antivenin. A negative skin test is no guarantee that an immediate reaction to antivenin will not occur, but a positive test (immediate wheal and flare reaction) will identify some of the sensitive patients. Patients with a markedly positive skin test or a history of allergy to horse serum should not be given antivenin unless the potential benefits are greater than the risk of systemic reaction to horse serum. Rapid desensitization of patients allergic to horse serum has been suggested but in practice is difficult and time-consuming, making it of questionable value. The bitten extremity ought to be comfortably immobilized in a neutral position. Laboratory tests should include complete blood and platelet counts, blood typing and cross-matching (this is often difficult when delayed), urinalysis, serum creatinine and BUN, and partial thromboplastin time. A dramatic fall in the platelet count is frequently seen with systemic poisoning, although hemorrhage in these cases is unusual. An electrocardiogram may be useful, particularly in older patients. An intravenous access should be maintained with a large gauge needle in the event that systemic poisoning and shock occur.

The amount of Antivenin (Crotalidae) Polyvalent to be given will depend on several factors, the most important being the severity of the symptoms and signs and their progression. Most authorities do not advise the use of antivenin for minor envenomations (defined as the presence of local swelling and pain that progresses slowly, if at all, over several hours, without evidence of systemic poisoning).[4,5,7-9] Antivenin is indicated in all cases where there is progressive local envenomation, paresthesias of the face and scalp or systemic poisoning. Progression of local envenomation can be monitored by measuring the circumference of the bitten part and two or more proximal sites. The measurements can be repeated every 15 minutes and serve as a guide for antivenin adminstration. In moderate envenomations, three to five 10 ml vials of antivenin should be administered initially in an intravenous drip of 0.9% NaCl over the first hour. The volume of saline used will depend on the fluid status, age and coexisting medical problems of the patient. An additional 3-5 vials should be given each hour until the progression of the signs and symptoms has been halted. In more severe local or systemic envenomations, 6 to 10 vials of antivenin should be given initially, and this amount repeated hourly until improvement occurs. Some adults have required 50 or more vials of antivenin for severe envenomation.

The most common errors in antivenin therapy are beginning therapy with too little antivenin or beginning therapy when it is not necessary. Serum sickness will occur about one to two weeks post-treatment in approximately 80% of patients receiving antivenin. This is usually a mild disease, characterized by fever, arthralgias, urticaria and malaise, that can be treated with aspirin, antihistamines and reassurance. More severe reactions reliably respond to a short course of glucocorticoids. Anaphylaxis from antivenin is extremely rare. However, because anaphylaxis can occur and the fact that antivenin is not heat stable, field use of antivenin is discouraged. The preferred route of Antivenin (Crotalidae) Polyvalent administration is intravenous.[5] Injecting some or all of the antivenin around the bite site can increase the edema and

compromise the blood supply in the area and would not be expected to produce useful blood levels. Intramuscular injection is associated with erratic absorption. The antivenin is most effective if given within four hours of the bite, but may be of value up to 24 hours post-bite.[10] In this author's experience and that of others[5,8,10,11] the antivenin is effective at neutralizing both the systemic and the local effects of injected rattlesnake venom.

The use of cryotherapy to treat snake venom poisoning, in the form of ice bags, ice slushes, and ice-cold water or compresses, is contraindicated. There is ample evidence in clinical studies that the majority of snakebite cases requiring amputation of an extremity are limited to those treated with chilling, insufficient antivenin or both.[3,7,9] Animal studies have confirmed that the damaging effects of injected snake venom are enhanced by cryotherapy, regardless of whether or not antivenin is administered.[11,12]

The routine use of fasciotomy to treat snake venom poisoning, as recommended by Glass,[13] is without medical rationale. Fasciotomy results in unnecessary disfigurement and tissue loss that in most instances can be prevented by the appropriate administration of antivenin. Russell[5] reports that fasciotomy has been unnecessary in his series of more than 650 cases of snake venom poisoning unless insufficient antivenin was given. Administration of glucocorticoids to treat snake venom poisoning is non-specific therapy and may impair the effectiveness of antivenin administered subsequently.

Before discharge the bite site should be inspected for signs of secondary infection. Prophlactic antibiotics are not advised. The appropriate antitetanus preparation should be given in all cases.

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OUT OF CONTEXT

The Medical School Scene

Doctor C. A. Hiebert voiced his concern about the medical school from which he graduated twenty-odd years ago. "Inasmuch as (this medical school) selects a first year class of dazzling genetic pool and controls student enviroment with extraordinary resources, what on earth happens to these promising young men and women between their first and fourth years?"

The Medical Expert Witness Scene

The following, in part, is from a letter of solicitation for expert medical "unbiased opinions."

". . .The Foundation a non-profit and tax exempt organization, that has many physicians on its consulting staff, who are willing to give honest and unbiased opinions to patients and attorneys and to support those opinions at depositions and in court. . ."

Some years ago, Doctor Lloyal Davis of Northwestern University Medical School stated it was his policy never to accept a medical-legal case for examination and review.

Furthermore, he would not appear in court, except in response to a suponea, and would not accept an expert witness fee.

My own experience was, after adopting this policy, I was suponed once, refused the "expert fee," and for the following 20 odd years was never again suponed in a personal injury case! So far as I know this did not interrupt "the flow of justice."

Your experience may be different, but I had previously noted, that when I did appear in court, which was frequent, no matter how "unbiased and factual" I believed my testimony to be--there was always a local colleague able to testify to the contrary. The jury or judge was then, "in their infinite wisdom," supposed to determine the true facts. Balderdash!

So to those of my medical peers who appear as an expert medical witness, "I have utmost faith in your infallability, but none whatever in my own." May your fee be comminsurate with the justice derived therefrom.

John W. Kennedy, M.D.

Arizona Medicine

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Editorials

AMA Board of Trustees Chairman to Visit Arizona

Lowell Steen, M.D., Chairman, Board of Trustees of AMA will be here in Tucson on Saturday, October 4. He will be coming as the American Medical Association's official representative, making the annual briefing to the component medical societies. Dr. Steen will be meeting with the officials of ArMA, county medical society presidents, representatives of state specialty societies and the faculty of the Medical School. That afternoon there will be a reception in his honor, at which time all of the physicians in the area can come and meet him and ask any question they have about AMA policies and activities

As a reminder, the Board of Directors will be meeting at 10 a.m. that same day (October 4) in Tucson.

AMA Meeting July 1980

In addition to the official Arizona Delegation attending the July AMA meeting in Chicago were your President-Elect, Clyde Kurtz, and his wife, as well as my wife and I, and ArMA staff people. Although neither Clyde nor I could vote, we did sit in on the deliberations of the delegates from the Rocky Mountain States Conference. The states comprising this conference are Arizona, Colorado, Idaho, Montana, Utah and Wyoming.

By going to these meetings and attending the reception, we had an opportunity to meet our counterparts in these other states. We also sat in on the official meetings of the House. This is a very exciting experience, and one that makes you proud to be a member of AMA. All in all, some 248 items of business were dealt with during the five day meeting. The whole process is very complex, and one can only fully appreciate this fact by being there in person. I would urge members to attend this meeting and also bring their wives. The Auxiliary meets concurrently. Spouses of ArMA's delegation may be designated to serve as an Auxiliary alternate, if they are members of their auxiliary.

The New President-Elect

By now you should know that Dan Cloud was elected President-Elect of AMA at this meeting. It's great to have a member so honored. Dan was the first Arizonan to ever seek the post, and his election culminated months of work by countless ArMA members in support of his candidacy.

Malpractice Insurance

During the past four years, the Mutual Insurance Company of Arizona (MICA) has been the only insurance company selling malpractice policies to all Arizona physicians. ArMA was instrumental in getting MICA started at the height of the malpractice insurance crisis. Since the crisis, state law requires that all carriers writing this type of insurance must provide coverage for any and all physicians wanting to buy it. Starting this October 1, the law will change, and carriers will once again be allowed to be selective about whom they will underwrite. Once again, it can be expected commercial carriers will soon enter this market on a selective basis. In order to develop this market, the carrier would probably offer low rates the first year, and then increase the rates in subsequent years. This was borne out by an article appearing in the *Arizona Daily Star* of July 30. The lead paragraph said:

"A Houston company is entering the Arizona medical malpractice insurance business with rates that apparently are too low to cover expected losses and could be loss leaders designed to destroy the only major insurer in the field, Arizona Insurance Director J. Michael Low said yesterday."

I am sure that Arizona physicians will remember who was here when the going got tough

John T. Clymer, M.D.
President - ArMA

Home Dialysis in Southern Arizona

Home dialysis is an important component of any complete program of renal replacement therapy. Home dialysis affords the patient with end-stage renal disease (ESRD) the opportunity to dialyze at a location and times convenient to himself or herself, and thereby permits the least possible interruption of lifestyle including work and leisure activities. In addition, it fosters an attitude of self-reliance rather than dependence on others, which maximizes the rehabilitation of patients with ESRD. Perhaps because of these effects, survival of patients on home dialysis has been shown to be better than those treated in dialysis facilities, better than those treated with cadaver transplantation, and equal to those treated with related donor transplantation. Home dialysis is also cost effective compared to any other form of renal replacement therapy with the possible exception of related donor transplantation.

A program of home dialysis, available to any area resident, was initiated at the Tucson Veterans Administration Medical Center in March 1972. In June 1978, a second program was initiated by the Dialysis Foundation of Southern Arizona, Inc., at its Artificial Kidney Center-East in Tucson, to meet the growing need for home dialysis training. These combined programs have trained 77 patients to perform home dialysis and have provided home dialysis support to an additional twelve patients trained at other centers. Seventy-four of these patients have come from eleven different Arizona counties; ten have come from other states; four have come from Mexico; and one has come from Canada. The 89 patients represent 19.4% of all patients entering our program of renal replacement therapy in southern Arizona.

Educationally, the program serves as an ideal example of the team model in medical care. A successful home dialysis program requires a dedicated and skilled home training team and provision of regular and emergency home support to every patient after completion of training and initiation of dialysis in the home. The home training team consists of the home training nurse, who also serves as the team leader; the home training technician, who is responsibile for teaching the machine and technical aspects of dialysis and for proper installation of necessary equipment in the home; the social worker, responsible for assessing the family strengths and needs with respect to home dialysis; the dietician, responsible for teaching dietary needs and restrictions to the home patient; and the patient's nephrologist, responsible for overall medical care, including the dialysis prescription, for the patient. Support of the patient after training includes regular and emergency visits to the home to check procedure, supplies, and equipment; retraining in new techniques as appropriate; and back-up center dialysis as necessary for patient illness, or helper vacation, illness, or simply temporary relief. The goal of the program is to keep the patient well and functional in the home setting as much of the time as possible.

In the past six months, both at the Veterans Admistration Medical Center and the Artificial Kidney Center-East, many of the new candidates for home dialysis have been trained in the new technique of continuous ambulatory peritoneal dialysis (CAPD) rather than the more conventional technique of hemodialysis. CAPD has the advantages of simplicity, in that no actual equipment other than an IV pole and accurate patient weighing scale are needed, and of mobility, since the procedure can quite literally be done anytime and anywhere with supplies that can be carried in a briefcase. It also, however, has the disadvantages of a constant risk of peritonitis with a documented incidence of about one episode every seven months, and peritoneal dialysate exchanges must be done four times a day seven days a week, every week without fail and with meticulous technique. Long-term acceptance and efficacy of this therapy remain to be proven, but through March 1980, seventeen patients in our program have been trained to perform CAPD at home.

Our home dialysis program is utilized by all nephrologists and is available to all ESRD patients in southern Arizona. The advent of new therapies and renewed emphasis and innovative funding by the federal government for home dialysis promises increased activity in this program in the future.

David A. Ogden, M.D.
Chief, Renal Section and Professor of Medicine
Department of Internal Medicine

Louis J. Kettel, M.D.
Dean, College of Medicine

FTC Broadens its Front in Attack on Medicine

To what degree will the Federal Trade Commission (FTC) be able to encroach on our medical professionalism and the public-spirited responsibilities that go with it?

This question loomed when the FTC challenged the AMA's ethical ban on physician solicitation of patients. It also is looming again on the issue of the FTC and medical-school accreditation, an issue that had been in abeyance during a two-year grace period.

The advisory committee to the U.S. Commissioner of Education is scheduled to open a hearing April 21 on the FTC's petition to dislodge the Liaison Committee on Medical Education (LCME) as accrediting body for medical schools.

As cosponsor of the LCME—along with the Association of American Colleges (AAMC)—the AMA is against the prime target of the FTC action, just as the ongoing case on physician advertising and solicitation.

FTC lawyers exultantly smell a rat in the AMA's accreditation role. They contend the Association wants to limit the number of schools and graduates for the same basic reason it allegedly wants to limit the scope of advertising; to stifle intraprofessional competition.

The contention is out of touch with the facts, of course. Instead of being held down, the number of approved medical schools has surged (with full AMA support) from 85 in 1960 to 126 today. Enrollment in that time has more than doubled.

Well, the FTC counters, the LCME just might sometime restrict the school growth.

Another federal agency—HEW—foresees no restriction. In a recent report, HEW states; "Our nation's schools are currently geared to produce doctors at a rate that should put the total number in balance with our national needs by the 1980s. By the 1990s, we should have a supply greater than we will need."

In its move on accreditation, the FTC is joined by such "public interest" groups as Common Cause and Sidney Wolfe's Public Citizen Health Research Group. But we feel it's clearly in the public interest that the AMA continues waging arduous if costly battles to keep medical professionalism intact.

American Medical Association

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Auxiliary Highlights

Theme for this 51st Auxiliary year is Facets of Communication and President Joyce Newman says the importance of good communication within the community will be stressed throughout the year.

"Good communication is the basis of any good relationship," continues Joyce. "Poor communication creates a multitude of problems wherever it occurs—hurt feelings, misunderstandings, confusion, arguements, a generation gap, broken friendships, even divorce. Much can be accomplished with good communication. Very little is accomplished with poor communication.

"We all communicate but what and how well we communicate is the important thing. To be a good speaker is not enough—we must be a good listener also. In fact, one probably is not a good speaker unless he is a good listener. This year the Auxiliary intends to become a better communicator and more avid listener.

"September 23-24 at the Fall Leadership Conference in Sedona, the First Facet of Communication will take place. Each and every Auxiliary member is invited to attend this meeting and see what the state and county organizations are doing and what is being planned to fulfill the state goals as outined in the summer issue of *Caduceus Crier*, the ArMAA newsletter. The Sedona conference is a time for communicating with Auxiliary members from around the state, for making new friends, for attending educational and leadership seminars. It's an opportune time to discover that our state and county Auxiliaries are viable, innovative, hardworking and influential forces in the community."

Conference Speakers

September Sedona Getaway—that's how President-Elect Janet Hoffman is billing the Fall Leadership Conference, which will be held September 23-24 at Poco Diablo resort in the red rock country. Committees will meet and the board will transact business but there will be special speakers and workshops, as well as time for relaxation and fun. Sally Hewitt of the Junior League, who has a master's degree in counseling, will speak on "Options," how to use volunteer hours and experience in entering the job market. Sara Aleman-Burns, M.S.W., a community service representative for the Department of Economic Security in Flagstaff and a former NAU faculty member, will discuss "Stress and Personal Values."

Mary Lee Collins will present the Washington State Medical Auxiliary's Cost Containment program, "The Cost of Caring." Doris Ong will show Auxilians how to use graphics effectively. Connie Wry and Faith Anderson will conduct a speakers' training workshop.

Members of the Coconino County Auxiliary will be hostesses at the picnic lunch the first day of the conference.

Ella Kimball Takes National Office

Ella Kimball of Phoenix was elected Western region vice-president of the American Medical Association Auxiliary at its annual convention in Chicago in July. Mary Ellen Vaughan of Washington was installed as president of the national Auxiliary and Isobel Dvorsky, a guest at ArMA's annual meeting in April, was named president-elect.

President Joyce Newman headed the Arizona delegation to the convention: First Vice-President Mary Lee Collins of Scottsdale, Second Vice-President Anita Rosenblatt of Kingman, State Treasurer Carolyn Morgan of Phoenix, Maricopa Auxiliary President Jan Lisboa of Phoenix, State Legislation Chairman Ruth Ogden of Tucson, and Past President Elosie Clymer of Tucson.

They participated in workshops on legislation, health projects, membership, AMA-ERF and a special session for treasurers; heard newscaster Jessica Savitch and PR consultant Letitia Baldridge speak; attended courses on Nutrition and "Facing Death—A Child's Point of View," listened to testimony at reference hearings before voting in the House of Delegates; and still had time to campaign for Dan Cloud in his successful race for the post of president-elect of the AMA.

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ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, June 27, 1980, a quorum being present, convened at 6:37 p.m., John T. Clymer, M.D., President and Chairman, presiding.

Department of Health Services

Dr. Dandoy reported on activities relating to the implementation of the optometric bill, the Crippled Children's Hospital and regulations relating to long-term care facilities.

Unified Membership

Implications of the April 25th House of Delegates' action retaining unified membership with the AMA was discussed at great length.

Mr. Jacobson reported on several options relating to Articles of Incorporation and/or Bylaws changes that could possibly be investigated.

It was moved and carried to recommend to the Board of Directors that they call a special meeting of the House of Delegates for the purpose of voting on the question of unified membership between the AMA and ArMA and that a special committee be immediately established to educate the delegates on all aspects of the matter.

Directors' & Officers' Liability Insurance

Mr. Robinson reported that bids have been sought on the subject of insurance.

Long Range Planning Committee

Dr. Oakley reported that no action has been taken to date on establishing this new committee.

Western Physicians Purchasing Association

Mr. Robinson reported on the 6/12/80 Board of Directors meeting of the WPPA. *No Action.*

Correspondence

Governor Babbitt's letters of June 12 and 14th acknowledging ArMA's recommendations regarding various appointments were received.

Other Business

Gary Barnett reported on the current status of the ICA proposals. *Received.*

FINANCE COMMITTEE

The meeting of the Finance Committee of the Arizona Medical Association, held at 5624 E. Grant Road, Tucson, Arizona, on July 9, 1980, convened at 7:12 p.m., Neopito L. Robles, M.D., Treasurer and Chairman, presiding.

Arizona Perinatal Trust

The committee reviewed the request of the Arizona Perinatal Program for funding assistance in the establishment of an Arizona Perinatal Trust and received the testimony of Harold D. Gabel, M.D., C. D. Christian, M.D. and Mr. Ted Warner in this matter. Considerable discussion ensued, after which the following action was taken.

It was moved and carried to recommend to the Board of Directors that, when the Arizona Perinatal Trust is legally established, the ArMA Foundation convey title to the furniture and equipment listed on Attachment "A" totaling $13,922.61; and further, when the trust is legally established, the Association will continue to provide accounting services at no charge to the trust for a three-year period not to exceed costs to ArMA of $30,000.00 during the three-year period with a provision for annual review.

Justification of Committee Budgets

The question of which committee chairmen should be asked to attend the fall meeting of the Finance Committee to justify their committee's individual budget was discussed.

It was moved and carried to ask all committee chairmen whose budget exceeds $1,000.00 to attend the fall meeting of the Finance Committee, except in the case of newly-appointed chairmen, all of whom are invited to the meeting irrespective of the size of their budget.

Scholarship Funds

The committee reviewed the Board of Directors' referral back to the committee its recommendation regarding the repeal of the resolution relating to scholarship funds.

It was moved and carried to recommend to the Board of Directors that it prepare a resolution which would make it possible for interest income funds not utilized by the scholarship program to revert to the general fund.

Financial Statement for Six Months Ending June 30, 1980

The committee reviewed in detail the financial statement for the six-month period ending July 30, 1980.

ARTICLES OF INCORPORATION AND BYLAWS COMMITTEE

The meeting of the Articles of Incorporation and Bylaws Committee of the Arizona Medical Association, Inc. held at 810 W. Bethany Home Road, Phoenix, Arizona, on Saturday, July 12, 1980, convened at 1:42 p.m., Charles E. Henderson, M.D., Chairman, presiding.

Bylaws Amendments Regarding Specialty Societies

The committee again considered the changes in the Bylaws to bring about representation of specialty societies in the House of Delegates, reviewing its proposed amendment prepared and revised subsequent to its May 31st meeting and, subsequent to further discussion and revision, took the following action:

It was moved and carried to recommend to the Board of Directors that, in order to effect the reorganization of ArMA's House of Delegates to include specialty society participation, the Bylaws of the Arizona Medicine Association, Inc. be amended as follows:

"CHAPTER VII
HOUSE OF DELEGATES

"Section 1. Composition of House; Meetings:—The House shall constitute the voting body of the Association and shall be composed of the elected Delegates of the county societies, ELECTED DELEGATES OF THE SPECIALTY SOCIETIES, elected Delegates of each duly constituted chapter of the American Medical Student Association in Arizona, elected Delegates of the duly constituted chapter of the Arizona Medical Association Housestaff Section, and members of the Board. Delegates who are thereafter elected as officers do not by such election lose their status as voting Delegates in the House. The past presidents of the Association shall be ex-officio members of the House of Delegates without the right to vote.

"The House shall meet at least once a year at the time of the Annual Meeting. In addition, special meetings of the House may be held at any time, upon at least six weeks' notice thereof to the Delegates, at the call of the Board, or upon the call of twenty Delegates (*representing at least eight*) WITH REPRESENTATION FROM AT LEAST SEVEN county societies.

"Section 2. Number of Delegates—

"(A) COUNTY SOCIETIES. Each county society shall be entitled to representation in the House by two Delegates or their alternates, for the first twenty active members or fraction thereof, and one additional Delegate for each additional twenty active members or major fraction thereof, as determined

on October first preceding the Annual Meeting.

"(B) SPECIALTY SOCIETIES. A STATE SPECIALTY SOCIETY SHALL BE ENTITLED TO REPRESENTATION IN THE HOUSE OF DELEGATES BY ONE DELEGATE AND ALTERNATE WHO SHALL BE MEMBERS OF THE ASSOCIATION IF (a) THE SOCIETY HAS A NATIONAL BOARD OF DIRECTORS RECOGNIZED BY THE AMERICAN BOARD OF MEDICAL SPECIALTIES, (b) A MINIMUM OF TWENTY MEMBERS PRACTICING IN ARIZONA, THE MAJORITY OF WHOM MUST BE MEMBERS OF THE ASSOCIATION, (c) THE ACTIVITY OF THE SPECIALTY SOCIETY IS MANIFESTED BY AN EXISTING ORGANIZATION OR STRUCTURE EXHIBITING A SLATE OF PERIODICALLY ELECTED OFFICERS, AN ESTABLISHED CONSTITUTION AND BYLAWS, AND A FREQUENCY OF MEETING AT LEAST ONCE A YEAR. IF THERE IS MORE THAN ONE QUALIFYING SOCIETY FOR A SPECIALTY, THE BOARD SHALL DETERMINE THE REPRESENTATIVE SOCIETY. A SPECIALTY SOCIETY SHALL HAVE AN ADDITIONAL DELEGATE AND ALTERNATE FOR EACH ADDITIONAL 150 MEMBERS OF THE SOCIETY WHO ARE MEMBERS OF THE ASSOCIATION, AS DETERMINED OCTOBER FIRST PRECEDING THE ANNUAL MEETING.

"(C) HOUSESTAFF SECTION; STUDENT ASSOCIATION. Each duly constituted chapter of the Arizona Medical Association Housestaff Section and each duly constituted chapter of the American Medical Student Association in Arizona shall be entitled to representation in the House by two Delegates or their alternates.

"Section 3. Election of Delegates; List Thereof: Sufficiently in advance of the Annual Meeting each county society, EACH SPECIALTY SOCIETY, The Arizona Medical Association Housestaff Section, and each duly constituted chapter of the American Student Medical Association in Arizona shall elect Delegates and an equal number of alternates to represent it in the House. In the absence of any Delegate, (*his*) THE alternate may vote in (*his*) THE DELEGATE'S name on any question before the House. No alternate shall be eligible for election to office, but (*he*) may be appointed to membership on House committees in the absence of the Delegate. The secretary of each county society, EACH SPECIALTY SOCIETY, (*and of*) the Arizona Medical Association Housestaff Section and each chapter of the American Medical Student Association shall send the list of such elected Delegates and alternates to the Secretary of the Association not later than two months before the Annual Meeting. Representation in the House shall be contingent upon compliance with this provision. A member to be seated must present evidence at the time of (*his*) appearance at the House (,) of (*his*) official election by (*his*) THE county society, SPECIALTY SOCIETY, (*or*) the Arizona Medical Association Housestaff Section, or chapter of the American Medical Student Association.

"Section 7. Quorum; Minimum Requirements to Vote. (*Fifteen*) TWENTY Delegates, or their alternates, (*representing*) WITH REPRESENTATION FROM seven county societies constitute a quorum, and unless otherwise specified herein, a majority vote of the quorum shall be required for action."

Establishment of Articles of Incorporation and Bylaws Committee as Standing Committee

The members discussed at great length the feeling that this committee should be discontinued as a special committee and established as a standing committee of the Association and took the following action:

It was moved and carried to recommend to the Board of Directors that the Articles of Incorporation and Bylaws Committee be established as a standing committee of the Association and Chapter VII, Section 4, of the Bylaws of the Arizona Medical Association be amended by the inclusion of the following:

(a) ARTICLES OF INCORPORATION AND BYLAWS:—THIS COMMITTEE SHALL CONSIST OF A CHAIRMAN AND AT LEAST NINE MEMBERS. ALL MEMBERS OF THIS COMMITTEE SHALL HAVE BEEN MEMBERS OF THE ASSOCIATION FOR AT LEAST FIVE YEARS PRIOR TO THEIR APPOINTMENT.

THE COMMITTEE SHALL RESPOND TO RESOLUTIONS OF THE HOUSE OF DELEGATES BY REPORTING ITS DELIBERATIVE OPINION AND/OR PREPARING PROPOSED AMENDMENTS TO THE ARTICLES OF INCORPORATION AND BYLAWS. THE COMMITTEE SHALL RESPOND TO DIRECTIVES BY THE BOARD BY REPORTING ITS DELIBERATIVE OPINION AND/OR PREPARING PROPOSED AMENDMENTS. ALL PROPOSED AMENDMENTS PREPARED IN RESPONSE TO HOUSE OR BOARD DIRECTIVES SHALL BE PRESENTED TO THE BOARD FOR ITS CONSIDERATION.

THIS COMMITTEE MAY (CHAPTER X, SECTION 1) PROPOSE AMENDMENTS FOR INTRODUCTION TO THE HOUSE OF DELEGATES.

Student Membership

The committee was apprised of certain problems which had arisen by reason of student membership in the Association and, while it was felt that consideration would and should be given to Bylaws changes which would allow for greater clarity of student organizations recognized by ArMA, the matter was tabled for the present.

Comings & Goings

Chemotherapy Trial Announced

Robert W. Shook
Program Director
Arizona Lung Association

Arizona physicians are being asked to cooperate in a double blind study of Ketoconazole in the treatment of pulmonary Coccidioidomycosis. Antonino Catanzaro, M.D. of the University of California Medical Center, San Diego, is the principal investigator. The Arizona Lung Association is cooperating by making the study known to Arizona physicians.

Ketoconazole is a new anti-fungal agent which provides good therapeutic levels when taken by mouth. It is effective against *C. immitis in vitro* and in a mouse animal model. Preliminary human studies are encouraging.

Patients with pulmonary Coccidioidomycosis who have been sick at least six (6) weeks, but who do not have other severe disease processes, high Complement Fixation titers, or other high risk factors will be included in this protocol. Patients will be placed either on Ketoconazole or placebo in a double-blind fashion. Treatment and followup will be continued for a least six months in each patient using the drug as a daily oral dose.

In general, the philosophy of this trial is to use Ketoconazole in those patients who ordinarily might be observed, rather than treated for their disease. It is to see if the clinical course of Coccidioidomycosis can be altered by this therapeutic agent.

Participating investigators in Arizona are listed below; they may be contacted directly regarding details of the study and referral of study patients.

Mesa:
Edward G. Foxley, M.D., Private Practice
834-9263

Phoenix:
Bernard E. Levine, M.D., Private Practice 258-4951

Jay Rohwedder, M.D., V.A. Medical Center 277-5551

Philip Rubin, M.D., Private Practice 253-6378

Tucson:
Barry Friedman, M.D., Private Practice 886-5463

John Galgiani, M.D., V.A. Medical Center, Arizona Health Sciences Center 792-1450, Ext. 424

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

SEPTEMBER

The Determination of Toxicity of Chemical Products.
September 16-17. Plaza International, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center of Occupational Safety and Health, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Hypertension Update - 1980
September 18, 1980, The Adams Hotel, Phoenix, Arizona. Sponsor: Good Samaritan Hospital, and Pfizer Laboratories. Contact:James Howard, M.D. (For professional information) (602) 257-4383 or Norma Bland (For other information) (602) 937-5578. Approved for 4 hours of Category 1 credit.

Dean's Clinical Rounds
September 18. U of A Health Sciences Center. Sponsor: U of A College of Medicine. Contact: Lolly Dawn, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Evaluation of Peripheral Neuropathy
September 24. Boswell Hosp. Rm. C119, Sun City. Sponsor and Contact: Martha R. Newby, Dir. of Education, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372.

Symposium Gastro-Intestinal Bleeding
September 26, 1980. Boswell Hospital Rooms 1-27, Sun City, AZ. Sponsor and Contact: Martha R. Newby, Dir. of Education, Walter O. Boswell memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for one hour of category 1 credit.

Comparative Techniques of Weight Control
September 26-27, Scottsdale Hilton Hotel, Scottsdale, AZ Sponsor: University of Arizona College of Medicine, and the Central Arizona District Dietetic Association. Contact: Janet Price, R.D., CADDA, 1051 S. Dobson Rd., No. 56, Mesa, AZ 85202. Approved for 10 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
September 26-27. Arizona Medical Assn. Offices. Sponsor: A.C.E.P., Arizona Affiliate, American Heart Assn., & ArMA. Contact: Carol Tuner, Arizona Medical Assn., 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

OCTOBER

Current Concepts In Cardio-Pulmonary Intensive Care
October 4, Phoenix Country Club, Phoenix, AZ. Sponsor: U. of A. College of Medicine, Doctor's Hospital, American Thoracic Society-Medical Section of the Arizona Lung Assn. Contact: Shorty Welnick, Medical Staff Services, Doctor's Hospital, 1947 E. Thomas Road, Phoenix, Arizona 85016. Approved for 8.25 hours of category 1 credit.

"Seeing Our Way Clear"
October 4, 1980. The Arizona Biltmore, Phoenix, Arizona. Sponsor: St. Luke's Hospital Medical Center. Contact: Chris Campbell, St. Luke's Hospital Medical Center. (602) 258-7373, Ext. 311. Approved for 6 hours of Category 1 credit.

Personal Instruction Course In Intra & Extra Capsular Cataract Extraction with IOL Implantation
October 8-11 and 22-25. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006.

Classical Aspects of Nutritional Support In the Community Hospital
October 11. Good Samaritan Hospital. Sponsor: Good Samaritan Hospital, Phoenix, Arizona. Contact: Good Samaritan Hospital Office of CME, 1033 E. McDowell Road, Phoenix, AZ 85006. Approved for 7 hours of category 1 credit.

Clinical Seminar Series "Pulmonary Diseases"
October 11. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724.

Second Annual Symposium on Infectious Disease and Antibiotic Update
October 11, 1980. Paradise Valley Country Club. Sponsor: St. Luke's Hospital Medical Center. Contact: Chris Campbell, Seminar Coordinator. St. Luke's Hospital Medical Center Phoenix, AZ. (602) 258-7373, Ext. 311. Approved for hour per hour Category 1 credit.

Pacific Coast Fertility Society Symposiums
October 16-19, 1980. Mountain Shadows Resort, Phoenix, Arizona. Sponsor: Pacific Coast Fertility Society. Contact: Dee Davis (213) 937-5514. Approved for 17 hours of category 1 credit.

Management of Common Neurologic & Neurosurgical Problems
October 17-18. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucosn, AZ 85724. Approved for hour per hour category 1 credit.

Practical Aspects of Infectious Disease in Obstetrics and Gynecology
October 18, 1980. U. of A. Health Sciences Center, Tucson, AZ. Sponsor: U of A Colelge of Medicine. Contact: Department of Obstetrics and Gynecology U of A Health Sciences Center, Tucson, AZ 85724.

Perinatal Update
Arizona Perinatal Trust
October 24-25, 1980. SunBurst Hotel, Scottsdale, AZ. Sponsor: Arizona Medical Association, Inc. Contact: Cathy Gunn, Arizona Perinatal Trust, 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 15 hours of category 1 credit.

Learning Disabilities: New Direction for the Physician
October 25, 1980. Sheraton-Scottsdale Inn, Scottsdale, Arizona. Sponsor: Good Samaritan Hospital. Contact: Mrs. Karen Iversen, Arizona ACLD Chairperson, 5211 E. Orchid Lane, Paradise Valley, AZ 85253. (602) 959-3192. Approved for 6 hours of category 1 credit.

Practical Management of Multiple Sclerosis
October 25, 1980. Phoenix, Baptist Hospital, Phoenix, AZ. Sponsor: Phoenix Baptist Hospital. Contact: Ruth Lee (602) 277-7441.

Neuro-Linguistic Programming
October 25 & 26, 1980. Aloha Hotel, Phoenix, Arizona Sponsor: Communication Dimensions, 1615 West Lewis, Phoenix, Arizona. Contact: Jeff Hatch, (602) 258-1702 or Judy Brieger (602) 993-2575.

7th Annual Fall Orthopedic Lectureship
October 30-November 1. Maricopa County General Hospital, Phoenix, Arizona. Sponsor: Maricopa County General Hospital. Contact: Department of CME, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008 Approved for 6 hours of category 1 credit.

NOVEMBER

Cardiology Symposium: Recognition & Management of Ventricular Arrythmias in Practice
November 1, 1980. Adams Hotel, Phoenix, Arizona. Sponsor: Maricopa County General Hospital, Dept. of Internal Medicine. Contact: Chairman, Dept. of Medicine, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008 (602) 267-5351. Approved for hour per hour category 1 credit.

A Course In Gynecological Laparoscopy
November 3-5, 1980. Good Samaritan Hospital, Auditorium, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: Office of CME, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ. Approved for 18 hours of category 1 credit.

Personal Instruction Course in Intra & Extra Capsular Cataract Extraction with IOL Implantation
November 5-8 and 19-22. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 19th Street, Ste 105, Phoenix, AZ 85006.

Clinical Seminar Series "Endocrinology"
November 8. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours of category 1 credit.

Diabetes Meilitus (Current Concepts)
November 12, 1980. Boswell Hospital, Room C-119, Sun City. Sponsor and Contact: Martha R. Newby, Dir. of Education, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for 1 hour of category 1 credit.

Medical Society of the United States and Mexico
November 12-15, Guadalajara, Jalisco, Mexico. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Exec. Secy., 3161 N. Pantano Road, Tucson, AZ 85715.

Dean's Clinical Rounds
November 13. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Lolly Dwan, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

American College of Physicians Regional Meeting
November 14-15, 1980. Arizona Biltmore Hotel, Phoenix, AZ. Sponsor: U of A College of Medicine. Contact: Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 9 hours of category 1 credit.

Arizona Pediatric Society
November 14-16. Westward Look Resort, Tucson. Sponsor: U of A College of Medicine & Arizona Pediatric Society. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 11 hours of category 1 credit.

"Risk Managment For Safety Professionals"
November 17. Student Union-U of A, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center for Occupational Safety and Health, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

Geriatrics Conference
November 20-21. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours category 1 credit.

The Legal Hurt of Chronic Pain
November 20, 21, 1980. The Mountain Shadows Resort, Phoenix, Arizona. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator. St. Luke's Hosptial, 525 N. 18th Street, Phoenix, AZ (602) 258-7373, Ext. 311. Approved for 14 hours of category 1 credit.

Comprehensive Review in Toxicology
November 20-22. Sheraton-Pueblo Inn, Tucson. Sponsor: Denver Institute of Clinical Toxicology. Contact: CRIT, c/o Peter Bryson, M.D, Emergency Department, Denver General Hospital, 8th Ave. & Cherokee St. Denver, CO 80204. Approved for 15 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
November 21-22, 1980. Arizona Medical Assn., Phoenix, AZ. Sponsor: ACEP, Arizona Affiliate-American Heart Assn., & ArMA. Contact: Carol Turner, ArMA, 810 West Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

DECEMBER

2nd Annual Sports Medicine Symposium
December 3-6. Marriott Hotel, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

International Congress on Ericksonia Approaches to Hypnosis and Psychotherapy.
December 4-8, Phoenix, Arizona. Sponsor: Maricopa County General Hospital and The Milton H. Erickson Foundation, Inc. Contact: Jeffrey K. Zeig, Ph.D. The Milton H. Erickson Foundation, Inc. 1935 East Aurelius Avenue, Phoenix, Arizona 85020. Approved for hour per hour category 1 credit.

Welding Safety and Health for the Mineral and Construction Industry
December 12, 1980. Marriott Hotel, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton. Arizona Center for Occupation Safety and Health, U of A Helath Sciences Center Tucson, AZ 85724. Approved for hour per hour category 1 credit.

1981
JANUARY

Eight Annual Symposium
Barrow Neurological Institute of St. Joseph's Hospital, & Medical Center - Controversies In Neurology and Neurosurgery
January 8-10, 1980. La Posada Resort, Scottsdale, Arizona. Sponsor: St. Joseph's Hosptial and Medical Center, and the Barrow Neurological Foundation. Contact: Richard A. Thompson, M.D., Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Clinical Recognition and Management of Heart Disease 1981
Twenty-Fourth Annual Scientific Session of the American Heart Association, Arizona Affiliate
January 21-24, 1981. University of Arizona College of Medicine, Main Auditorium, Tucson, AZ. Sponsor: University of Arizona College of Medicine, American College of Physicians and the Arizona Heart Association. Contact: Office of CME and Outreach U. of A. Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

FEBRUARY

Seventh Annual Frontiers In Ophthalmology
February 12-14, 1981. The Arizona Biltmore Resort. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator, St. Luke's Hospital. 525 N. 18th St., Phoenix, Arizona (602) 258-7373, Ext. 311. Approved for 15 hours of category 1 credit.

Third Annual Practical Counseling Technique Workshop for the primary Physician
February 14-21, 1981. Lion Square Lodge, Vail, Colorado. Sponsors: Institute for Human Relationship and Development and Colorado Academy of Family Physicians. Contact: John C. Faul, M.D., 5290 E. Yale Circle, Ste 200, Denver, Co. 80222.

Third Annual International Catarct Surgery Symposium
February 16-18, 1981. La Posada Resort Hotel, Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

Ninth Annual Taos Lung disease Symposium
February 20-22, 1981. Holiday Inn, Taos, New Mexico. Sponsor: New Mexico Chapter of the American Thoracic Society. Contact: New Mexico Chapter of the American Thoracic Society, 216 Truman, NE, Albuquerque, New Mexico 87108.

Fourth Annual Mid-Winter Symposium in OB/GYN
February 25-27, 1981. Scottsdale Hilton Hotel, Scottsdale, Arizona. Sponsor: Maricopa County General Hospital, Dept. of OB/GYN, and Phoenix OB/GYN Society. Contact: Secretary, Phoenix OB/GYN Society, 333 E. Virginia, Ste 222, Phoenix, AZ 85004. (602) 257-9218.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.
Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.
A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.
Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.
Neurosurgical Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third and 5th, Eighth Floor Conference Room.
Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.
Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists
Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.
Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.
Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.
Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.
Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.
Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.
Tumor Board
last Thursday of the month, 12 Noon, Educ. Center Classroom I & II, May, July, Sept, & Nov.
Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II. Contact: Brian Updegraff, M.D.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.
Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.
CME Programs
Weekly Tuesday, 7 p.m., Cafeteria-West End.
Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.
Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

DOCTORS HOSPITAL

1747 East Thomas Road, Phoenix, Arizona 86016 Contact: Medical Staff Secretary for additional information.

Physicians Continuing Education Program
1st Thursday, 12:30 p.m., Classrooms.

ELDORADO HOSPITAL TUCSON (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.
Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.

Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm

Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.

Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.

Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.

Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North

Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.

Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 A.M., Contact: Paul Bergeson, M.D.

Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.

Clinical Conferences
2nd & 4th Wed., Maryvale Samaritan Hospital, Med. Staff Lounge

Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.

Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.

Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.

Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.

OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.

PM & R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.

Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.

GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.

Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora

Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.

Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology

Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.

Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.

Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.

Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.

Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.

PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: J. Goldberger, M.D.

Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman

Ophthalmology Conference
1st Monday, 12:30 P.M., Contact: Bruce Gillers, M.D.

Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.

Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D.

Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: B. Phibbs, M.D.

Clinical Pharmacology Conference
5th Tuesday, 12:30 P.M., Contact: M. Katz, Clinical Pharm.

12Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact J. Laguna, M.D.

Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: M. Baker, M.D.

Medical Surgical Grand Rounds
2nd Wed., 12:30 P.M., Contact: Chief Medical Resident

Medical Conference
1st Wed, 12:30 P.M., Contact: Chief Medical Resident

Cardiology Conference
1st Thursday, 12:30 P.M., Contact: B. Phibbs, M.D.

Endocrine Conference
2nd Thursday, 12:30 P.M., Contact E. Munjack, M.D.

GI Conference
4th Thursday, 12:30 P.M., Contact: C. Kanun, M.D.

Mortality & Morbidity Conference
3rd. Thursday, 12:30 P.M., Contact: E. Munjack, M.D.

OB/GYN Dept. Meeting
2nd Thursday, 2 P.M., Contact J. Wurzel, M.D.

OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: J Wurzel, M.D.

OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 P.M., Contact: J. Wurzel, M.D.

Clinical Pathology
5th Thursday, 12:30 P.M., Contact: J. Fleishman, M.D.

Renal Conference
1st Friday, 12:30 P.M., Contact: T. Pence, M.D.

Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D.

Rheumatology Conference
3rd Friday, 12:30 P.M., Cntact J. Goldberger, M.D.

Chest Conference
5th Friday, 12:30 P.M., Contct: D. Engelsberg, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008
Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

Clinical Conferences
2nd & 4th Wednesday, 12-1 p.m., Medical Staff Lounge

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 85008. Contact: Leonard Tamsky, M.D.

Medicine Conference
Daily 12-1 P.M., Contact: S. Schaffner, M.D.

Chest Surgery Conference
Weekly, Mondays, 1:30 P.M., Santa Cruz Rm.

Hepatology Conference
2nd Tuesday, 1:00 P.M., Contact: Walt Koppenbrink, M.D.

OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Room

Obstetrical Problem Conference
Weekly Tuesday, 7:30 A.M., Yavapai Rm.

Orthopedic Conferencé
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.

Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.,Contact: Robert Ganelin, M.D.

Urology Discharge Planning Conference
Weekly, 11:30 A.M., Station 42

Hand Surgery Conference
Weekly Wed, A.M., Santa Cruz Rm.

Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.

OB/Neonatology Seminar
Weekly, Wed. 7:30 A.M., Yavapai Rm.

Clinical Psychiatric Conference
Weekly, Wed., 11-12 P.M. Mental Health Annex, Rm. 1346.

Surgery Conference
Weekly Wed., 7-8 A.M., Surgical Dept.

Cardiology Conference
Weekly Thurs., 2 P.M., Santa Cruz Rm.

OB/GYN Resident Conference
Weekly Thurs., 12 P.M., Yavapai Rm.

GYN Endocrine Seminar
1st & 3rd Friday, 12:30 P.M., Sanata Cruz Rm.

OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 A.M., Yavapai Rm.

Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & HEALTH CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, Wed. and Friday, 12 Noon

OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conf. Rm.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for Category 1 credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Contact: D. Randall, M.D.

Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 A.M.

Otolaryngology Grand Rounds
4th Wednesday, 4-5 p.m., Conference Rm. A, Contact: N. Wendell Tood, M.D.
Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 A.M., Conf. Rm. A, Contact: Drs. L. Fairbanks & E.Y. Hooper.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for Category 1 credit.
Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Clinical Conferences
Weekly Tuesday, 12 Noon, Kiva Conference Room
Tumor Board Conference
Weekly Friday, 12 P.M., Kiva Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.
Family Practice Conference
1st Monday, 12:30 P.M., Doctors' Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiolpulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs, 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical—Surgical Rdiological Conf.
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.
OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Genetics Conference
Weekly Monday, 12:30 P.M., Pediatric Department
Pediatric Rounds
Weekly Monday, Wed., & Friday, 10:30 A.M., Pediatric Department
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.
ECG. Conference
Weekly Tuesday, 12:30 P.M., Pediatric Department
Medical Grand Rounds
Weekly Wednesday, 8 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 8 A.M., PIMC
Visiting Professor Informal Rounds
Weekly Thursday, 9:30 A.M., 1st Floor Conf. Room
Visiting Professor Formal Presentation
Weekly Thursday, 12:30 P.M., PIMC.
Nephrology Conference
Weekly Friday, 12:30 P.M., Pediatric Department

ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N. Wilmot Road, Tucson, AZ. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Family Practice Department Meeting
3rd Tuesday, 12 P.M., Contact Wm. Moteforte
Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 P.M., Contact: Kim Sowards.
Current Concepts in Medicine
Weekly Tuesday, 12 P.M., Auditorium.
Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 P.M., Contact: S. Salmon, M.D.

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.
Cardiac Conference
Weekly Monday, 12:15 P.M., Auditorium
Surgery Conference
1st Tuesday, 12:15 P.M., Auditorium
Emergency Medicine Conference
1st Wednesday, 12:15 P.M., Auditorium
Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 P.M., Auditorium
Psychiatry Conference
3rd Thursday, 7 A.M., Auditorium
Combined Medical-General Practice Conference
1st Friday, 12:15 P.M., Auditorium
Toxicology Grand Rounds, Auditorium
2nd Friday, 7:30 A.M., Auditorium
Ophthalmology Conference
1st Saturday, 8:30 A.M., Auditorium.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Emergency Medicine Lectures
Weekly Thursday, 8 A.M., Century Room A
Mental Health Update
1st Friday, 11:30-1:00 P.M., Century Room A
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.
Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact: Drs. Otto & Zehngut.
Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 P.M., HSC Auditorium, Contact: Cancer Center
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Room 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.
Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn
Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.
Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.
Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford
Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room
Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross
Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.
Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley
Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.
Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.
Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital
Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.
Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.
Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson
Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655
Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich
Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.
Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology
Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.
Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.
Resident's Conference
Wednesday, weekly 5-6 p.m., Diag Radiology Conf. Rm.
Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.
Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.
Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.
Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.
Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.
Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.
General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Cardiology Conference
Weekly Monday, except last, 12 Noon, Contact: J. Fernadez, M.D.
Dermatology Conference
4th Monday, 5:00 P.M., Contact: R. Miller, M.D.
Endocrinology Conference
Last Monday, 12 P.M., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.
Chest Conference
3rd Tuesday, 12 Noon, Contact: William Nevin, M.D.
Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Infectious Disease Conference
2nd Tuesday, 12 Noon, Contact: B. Friedman, M.D.
Nephrology Conference
1st Tuesday, 12 Noon, Contact: Dr. Seltzer.
Orthopedic Conference
1st Tuesday, 8:00 P.M., Contact: Jay Katz, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 P.M., Contact: Dr. Lightner.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.
Clinical Pathology Conference
Last Wednesday, 8:00 A.M., Contact: Dr. Fuchs.
Family Practice Meeting
2nd Wednesday, 12:30 P.M., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.
Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buschsbaum, M.D.
Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.
Tumor Conference
Weekly Thursday, 12 Noon, Contact: Dr. Hastings.
G—I Conference
Weekly Friday, 12 Noon, Contact: C. Krone, M.D.
Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.
OBGYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.
OBGYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.
Surgical Department Confernece
Weekly Saturday, 7:30 A.M., Contact: Dr. Hastings, TMC.
Surgical Grand Rounds
Weekly Saturday, 9:00 A.M., UAHSC-Room 5403, Contact: Dr. Norton.

PHOENIX VETERANS ADMINISTRATION MEDICAL CENTER

7th Street and Indian School Road, Phoenix, AZ 85012. Contact: Alfred Heibrumn, M.D. Approved for Category 1 credit.
GI X-Ray Clinical Correlation Conf.
1st & 3rd Monday, 1 P.M., Rm. C435, Contact: Drs. Sanowski, & Grobe, Ext 618.
Medical/Surgical GI Conference
1st Monday, 3 P.M., Rm. 3134, Contact: Dr. Kozarek, Ext 413. Dr. Mertz, Ext 493.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 P.M., RM. 3134, Contact: Dr. Mertz, Ext 493.
GI Pathology Conf.
2nd & 4th Monday, 1 P.M., Path Conf. Rm, Contact: Dr. Sakowski.
Vascular Conference
2nd Monday, 3 P.M., Room 3134, Dr. Cintora, Ext 419
Surgery/Pathology Conference & Tissue Review
1st & 3rd Tuesday, 4 P.M., Room 2128, Contact: Dr. Landard
Surgery Mortality/Morbidity Conference
4th Tuesday, 4 P.M., Room 3134, Contact: Dr. G. Mertz.
GI Grand Rounds
Weekly Tuesday, 1 P.M., Contact: Drs. Sanowski, Kozarek & Partyka
Cancer Symposium
Weekly Wed., 1 P.M., T-5, Contact: Dr. Byrne, Ext. 426.
Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 P.M., Contact: Dr. Rohwedder, Ext. 388.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 P.M., Rm 3134 Contact: Dr. Mertz, Ext. 493.
Medical Surgical Chest Conference
1st & 3rd Thursday, Contact: Dr. Rohwedder or Dr. Gill.
Cardiology Conference
2nd Thursday, 1 P.M., Rm. T-5.
Medical Service Grand Rounds
Weekly Friday, 11 A.M., Contact: Dr. Zeller
Surgical Journal Review & Videotape Session
Weekly 1 P.M., Room 3134.

PRESCOTT VETERANS ADMINISTRATION HOSPTIAL MEDICAL CENTER

Prescott, Arizona 86313. Contacts listed below. Approved for category 1 credit.

Medical Lectures
1st & 2nd Thursday & Weekly,Fridays, 1:30 P.M., Bldg. 28. Contact: J. W. Fitzharris, D.O.

GI Rounds
Third Thursday, 10 A.M.-2:30 P.M., Contact: J. W. Fitzharris, D.O.

Medical Rounds
First & seond Thursday, 1:30 P.M., Bldg. 28, Contact: J. W. Fitzharris, D.O.

TUCSON VETERANS ADMINISTRATION HOSPITAL & MEDICAL CENTER (U of A Tucson)

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2 P.M., Contact: Dr. Young

Medical Grand Rounds
Weekly Wed., 12-1:00 P.M., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

Grand Rounds
Weekly, Thursday, 11 A.M., Bldg. 107, Contact J. Fitzharris, D.O.

Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon.

Neurology Grand Rounds
Weekly Friday, 12 P.M., Contact: Dr. Sibley.

YUMA REGIONAL MEDICAL CENTER (U of A, Tucson/ArMA)

2400 Avenue A., Yuma Az 85364. Contact:Alan Winfield, M.D. Approved for category 1 credit.

Radiology Conference
1st Tuesday, 7:00 A.M.

Operation Outreach
2nd Tuesday, 6:30 P.M.

Pathology Conference
4th Tuesday, 7:00 A.M.

Operation Outreach
2nd Wednesday, 7:00 A.M.



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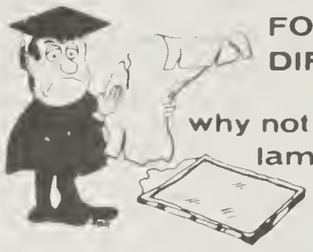
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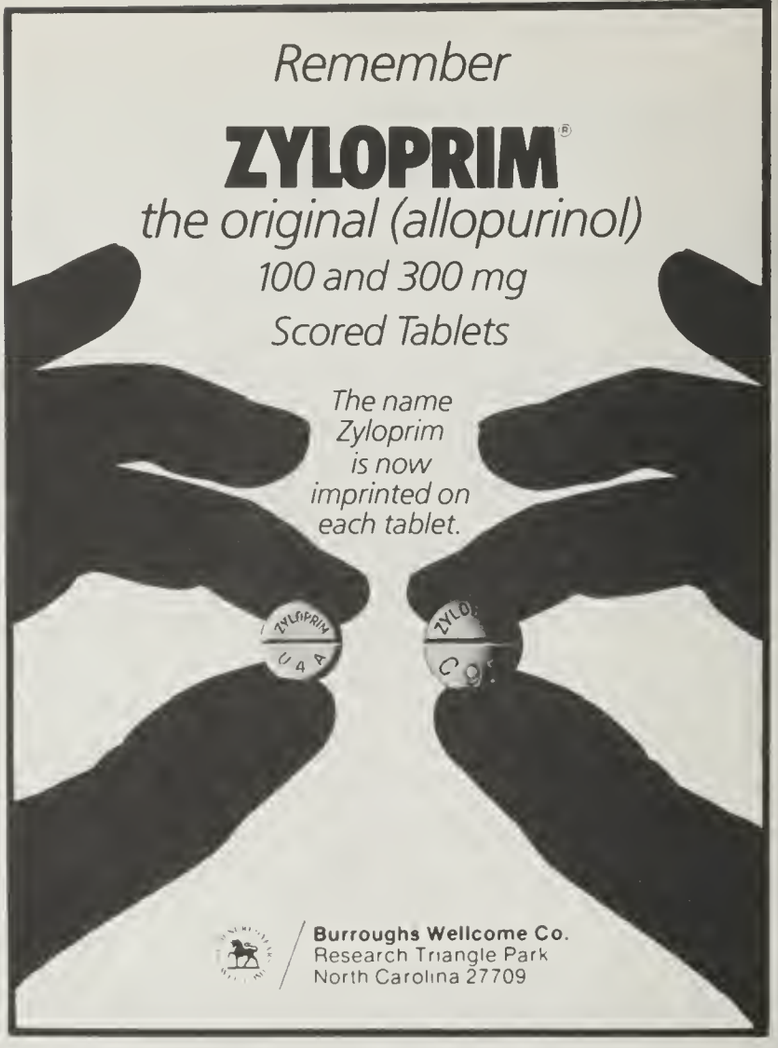
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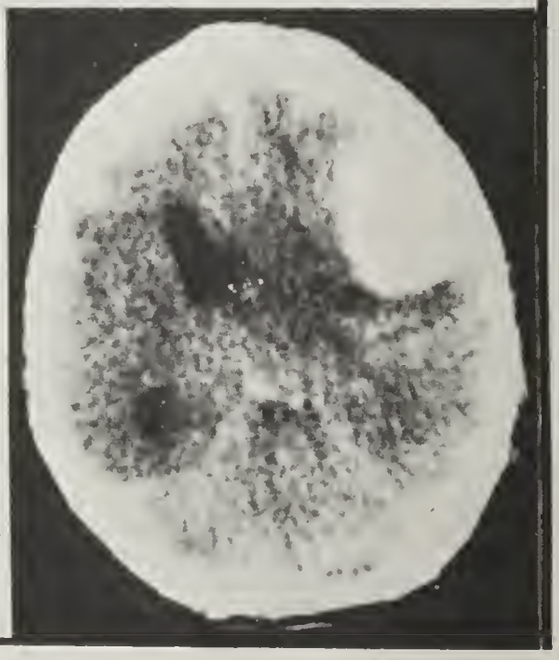
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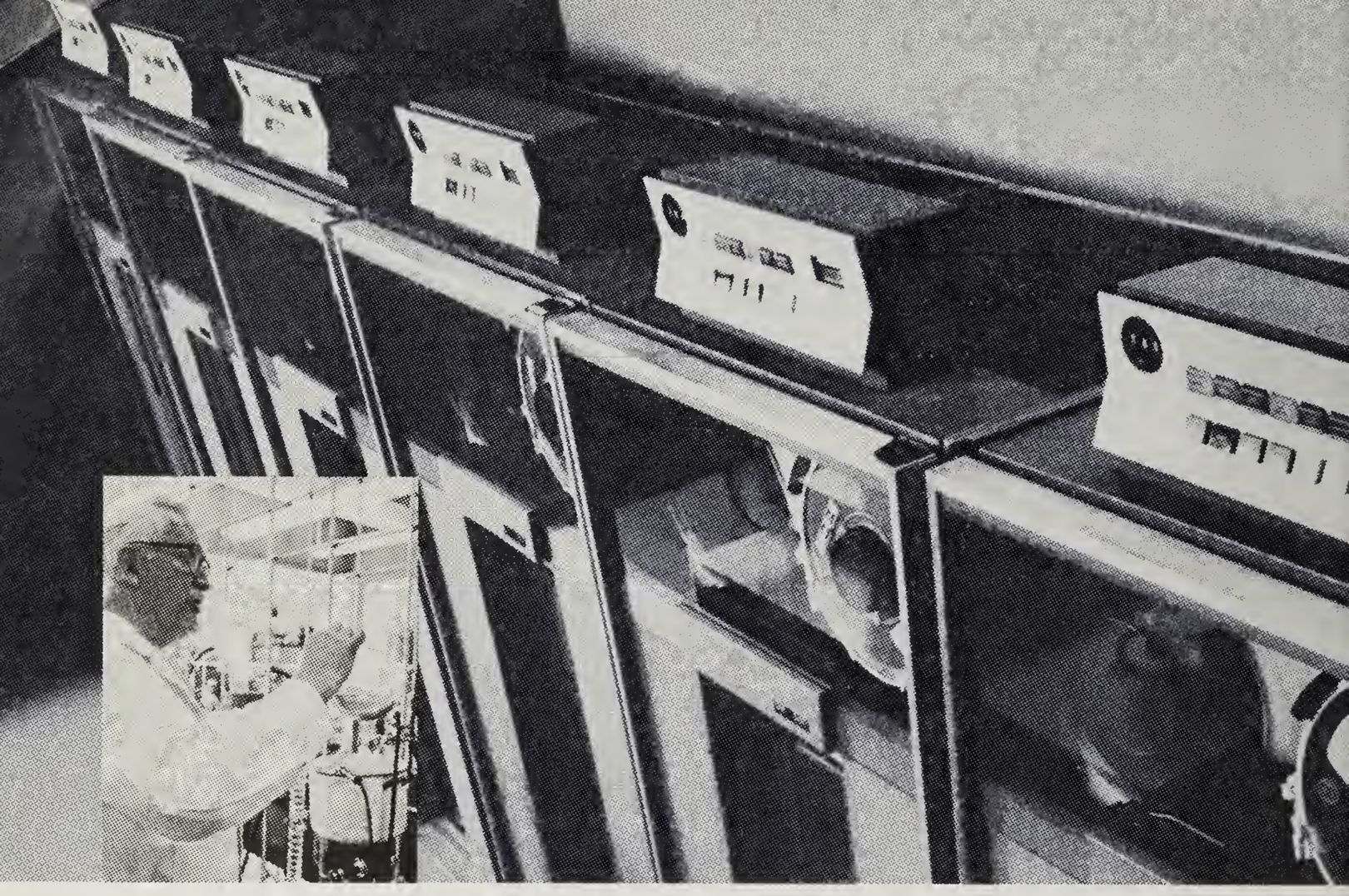
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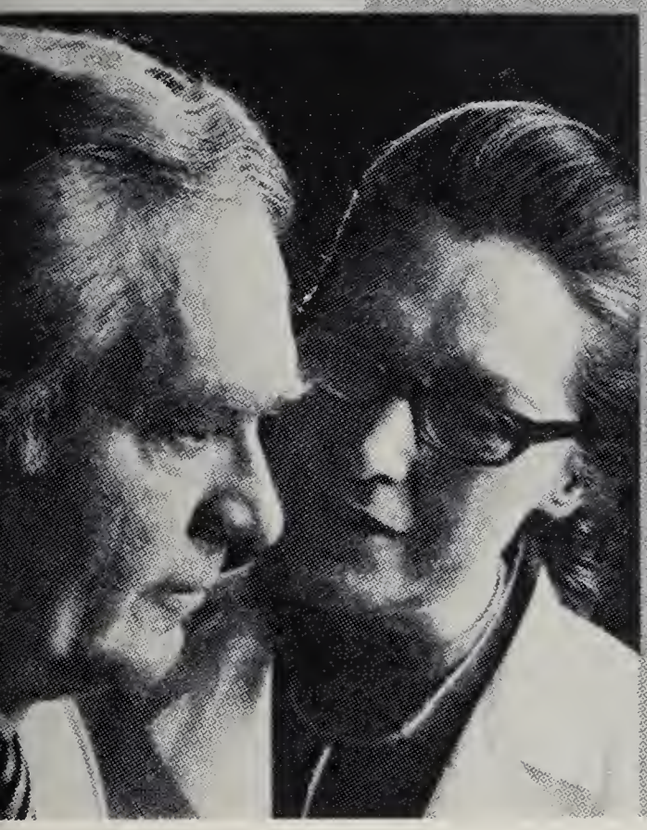
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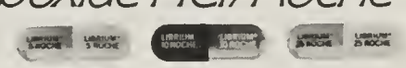


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chlordiazepoxide HCl/Roche

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended

Contraindications: Patients with known hypersensitivity to the drug

Warnings: Warn patients that mental and or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

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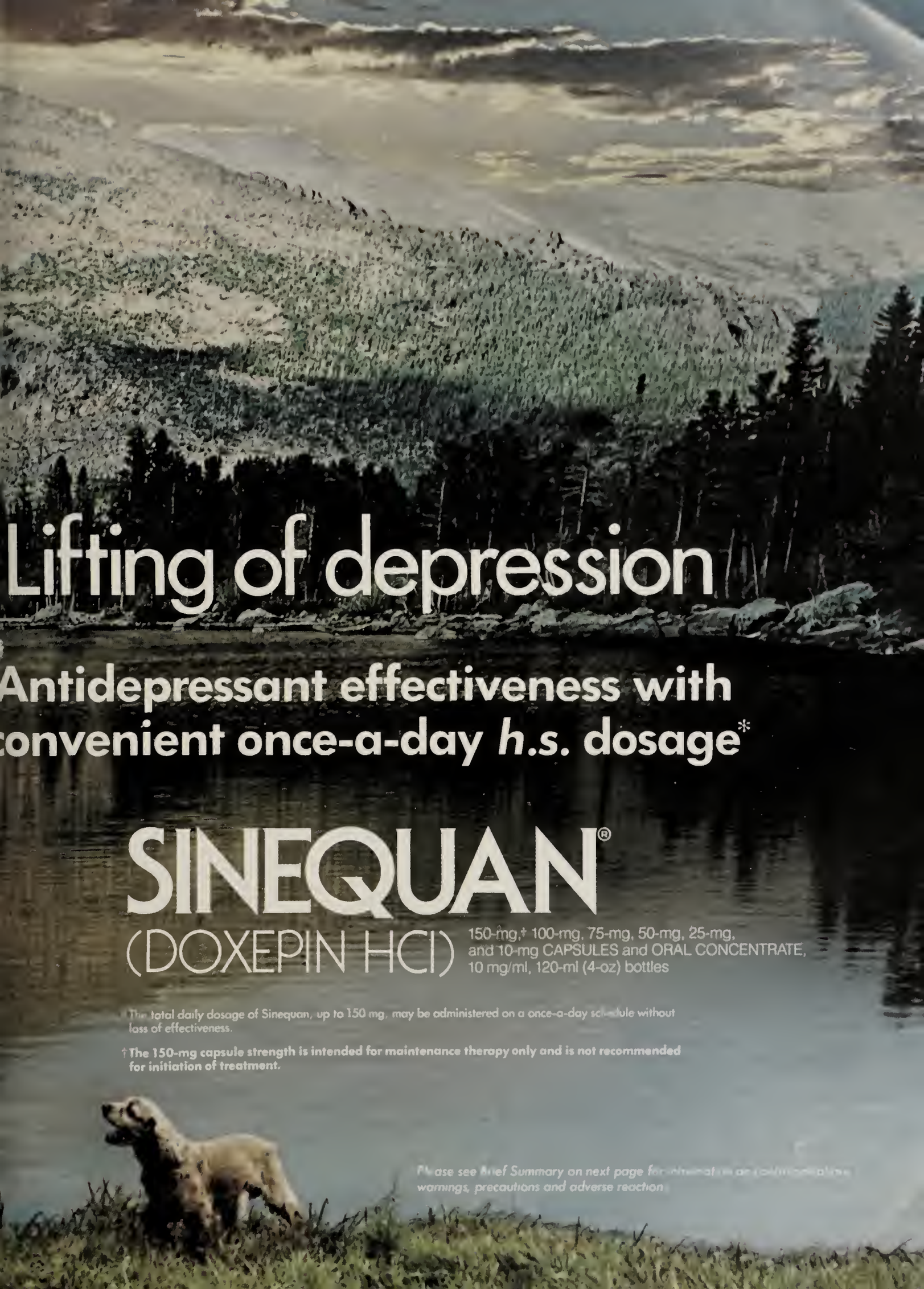
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†The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.
Please see Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. SINEQUAN is contraindicated in individuals who have shown hypersensitivi to the drug. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

SINEQUAN is contraindicated in patients with glaucoma or a tendency to urinary retentio These disorders should be ruled out, particularly in older patients.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness patients taking other medications should be carefully adjusted. This is especially important patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatr patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies have been performed in rats, rabbits, monkey and dogs and there was no evidence of harm to the animal fetus. The relevance to humans is n known. Since there is no experience in pregnant women who have received this drug, safety pregnancy has not been established. There are no data with respect to the secretion of the drug human milk and its effect on the nursing infant.

Usage in Children: The use of SINEQUAN in children under 12 years of age is not recom mended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following th concomitant use of certain drugs with MAQ inhibitors. Therefore, MAQ inhibitors should b discontinued at least two weeks prior to the cautious initiation of therapy with SINEQUAN. Th exact length of time may vary and is dependent upon the particular MAQ inhibitor being used, th length of time it has been administered, and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the dange inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned the possibility and cautioned against driving a car or operating dangerous machinery whil taking the drug. Patients should also be cautioned that their response to alcohol may b potentiated.

Since suicide is an inherent risk in any depressed patient and may remain so until significa improvement has occurred, patients should be closely supervised during the early course therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may b necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not bee specifically reported with SINEQUAN use. However, due to the close pharmacological similaritie among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects. Dry mouth, blurred vision, constipation, and urinary retention hav been reported. If they do not subside with continued therapy, or become severe, it may b necessary to reduce the dosage.

Central Nervous System Effects. Drowsiness is the most commonly noticed side effect. Thi tends to disappear as therapy is continued. Other infrequently reported CNS side effects ar confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramida symptoms and seizures.

Cardiovascular. Cardiovascular effects including hypotension and tachycardia have bee reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasiona reports of bone marrow depression manifesting as agranulocytosis, leukopenia, throm bocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, an aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargemen of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have bee reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a startin daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased a appropriate intervals and according to individual response. The usual optimum dose range is 7 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase t 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 15 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended fo maintenance therapy only and is not recommended for initiation of treatment.**

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effec may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.

2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to 10 mg, 75 mg and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade,® lemonade, orange juice, sugar water, Tang,® or water, but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

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(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence.* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy:* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children:* Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.

ADVERSE REACTIONS: *Cardiovascular:* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System:* Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal:* Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic:* Urticaria, rash, ecchymosis, erythema. *Endocrine:* Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System:* Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous:* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release. One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M.. A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S. Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp. 391-404.

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In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

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The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

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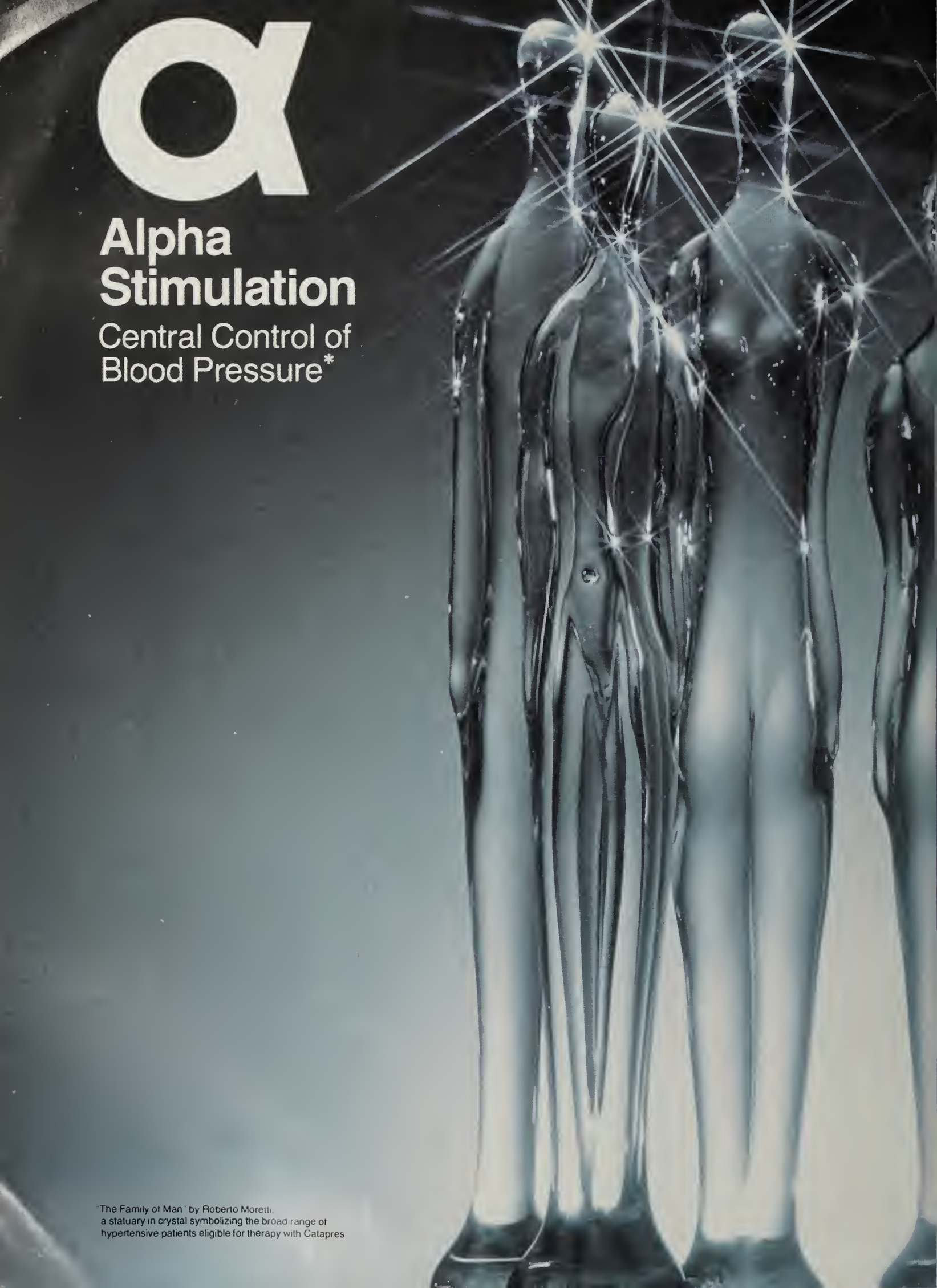
*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

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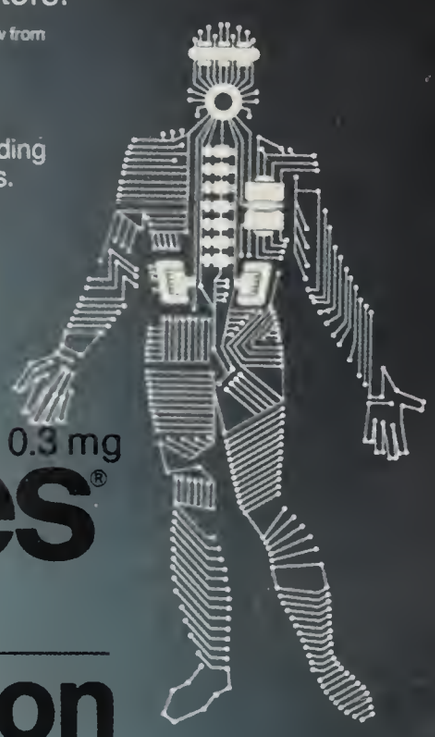
Other factors must include:

The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

1 Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.



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- Preserves kidney blood flow.

Most common side effects are dry mouth, drowsiness, and sedation which generally tend to diminish with time.

The usual starting dose of Catapres is 0.1 mg at breakfast and 0.1 mg at bedtime. Some patients may benefit from a starting dose of 0.1 mg at bedtime.

Usual daily dose range—0.2—0.8 mg

Maximum daily dose—2.4 mg
Doses as high as this have rarely been employed.

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Indication: The drug is indicated in the treatment of hypertension. As an antihypertensive drug, Catapres (clonidine hydrochloride) is mild to moderate in potency. It may be employed in a general treatment program with a diuretic and/or other antihypertensive agents as needed for proper patient response.

Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness a sedation. Constipation, dizziness, headache, and fatigue have been report Generally these effects tend to diminish with continued therapy. The followi reactions have been associated with the drug, some of them rarely. (In so instances an exact causal relationship has not been established.) These includ Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities liver function tests; one report of possible drug-induced hepatitis without icteru and hyperbilirubinemia in a patient receiving clonidine hydrochloride, chlo thalidone and papaverine hydrochloride. Weight gain, transient elevation of bloo glucose, or serum creatine phosphokinase: congestive heart failure, Raynaud phenomenon; vivid dreams or nightmares, insomnia, other behavioral change nervousness, restlessness, anxiety and mental depression. Also rash, a gioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associate with a rash, impotence, urinary retention, increased sensitivity to alcohol, drynes itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomasti weakly positive Coombs' test, asymptomatic electrocardiographic abnormalitie manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or ab sent reflexes and vomiting followed the accidental ingestion of Catapres (clonidin hydrochloride) by several children from 19 months to 5 years of age. Gastr lavage and administration of an analeptic and vasopressor led to complete r covery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-minut intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) ove dosage.

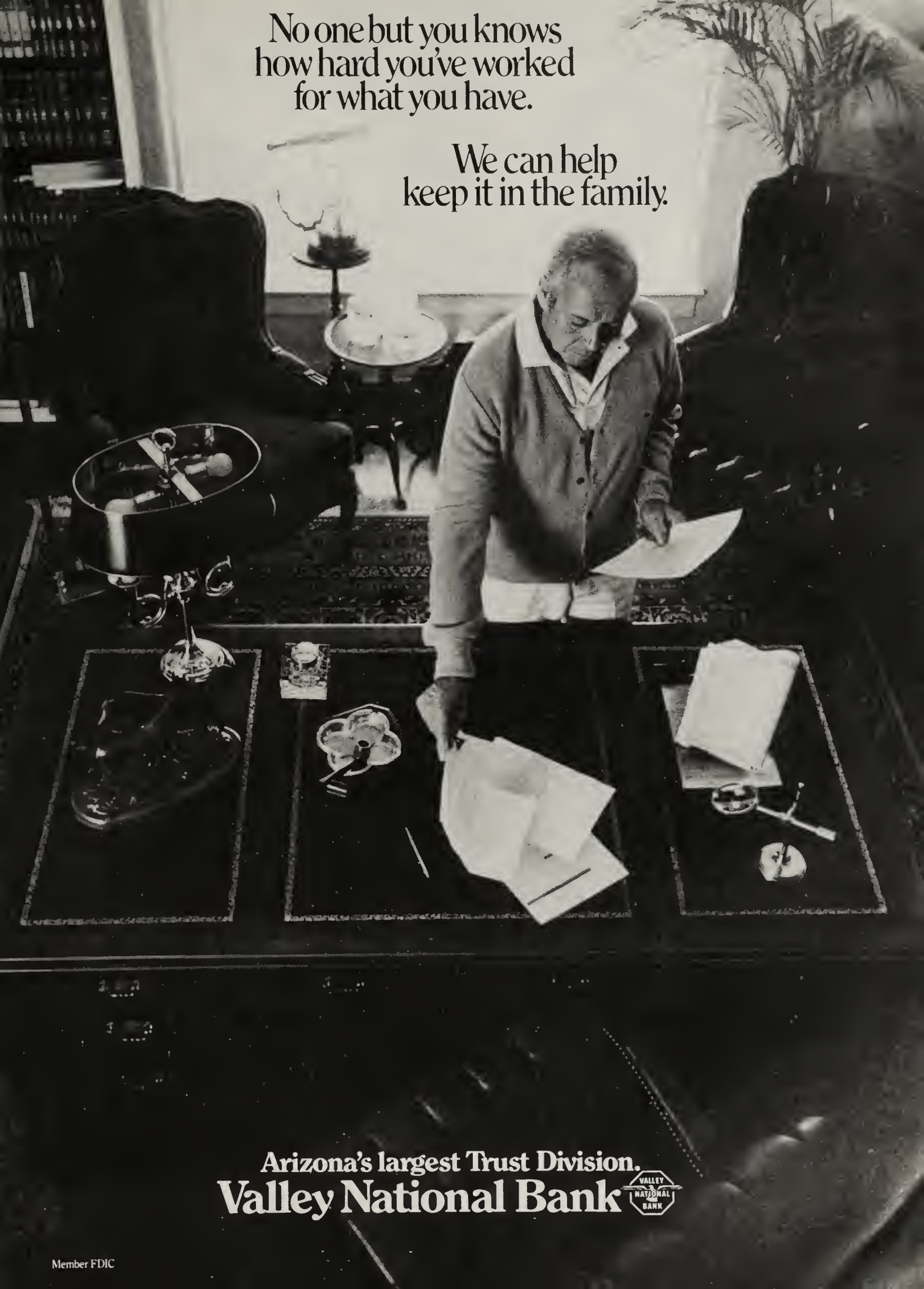
How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 m (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Als available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

For complete details, please see full prescribing information.
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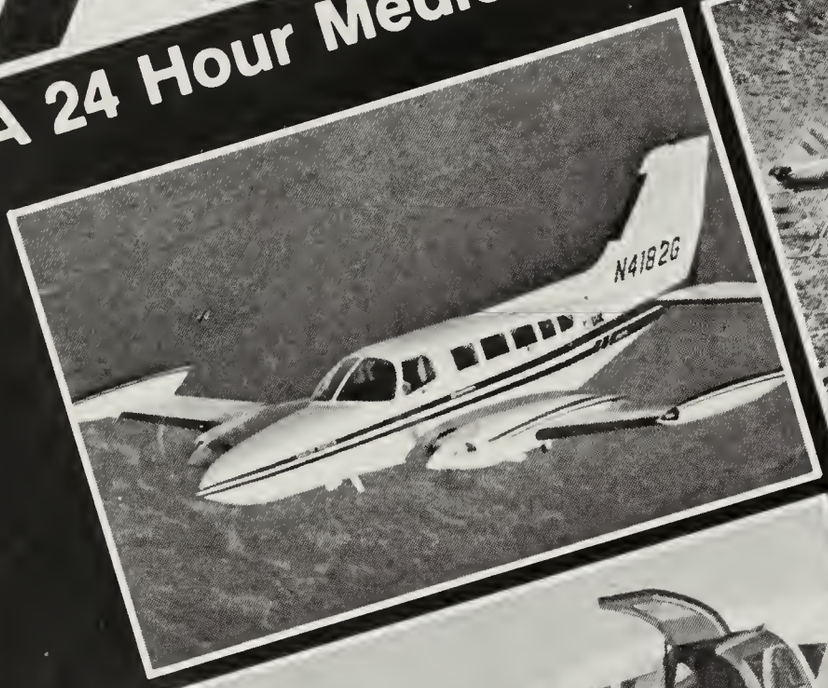
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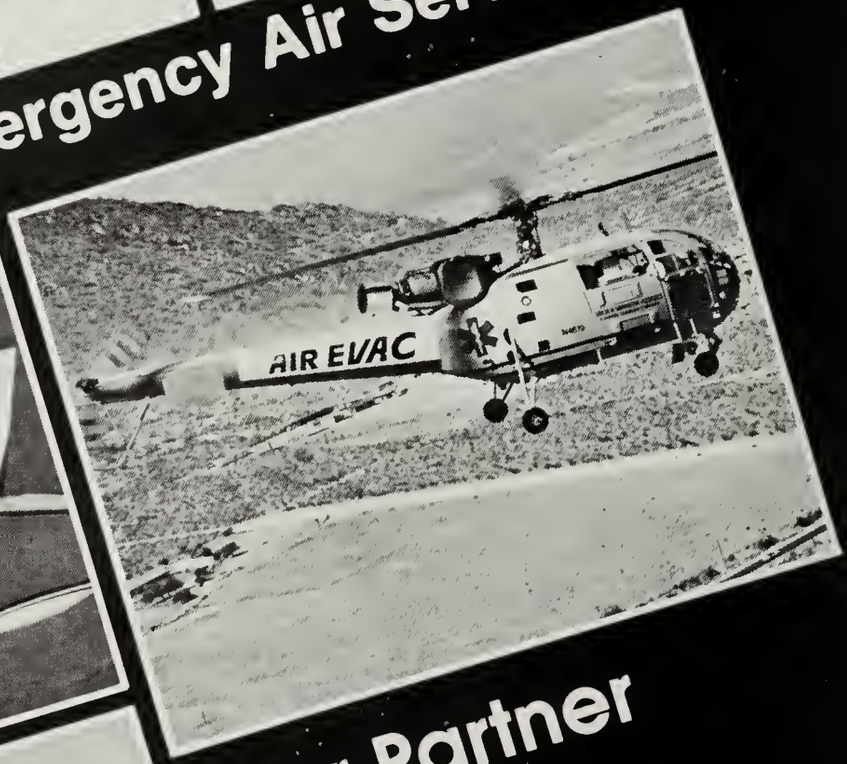

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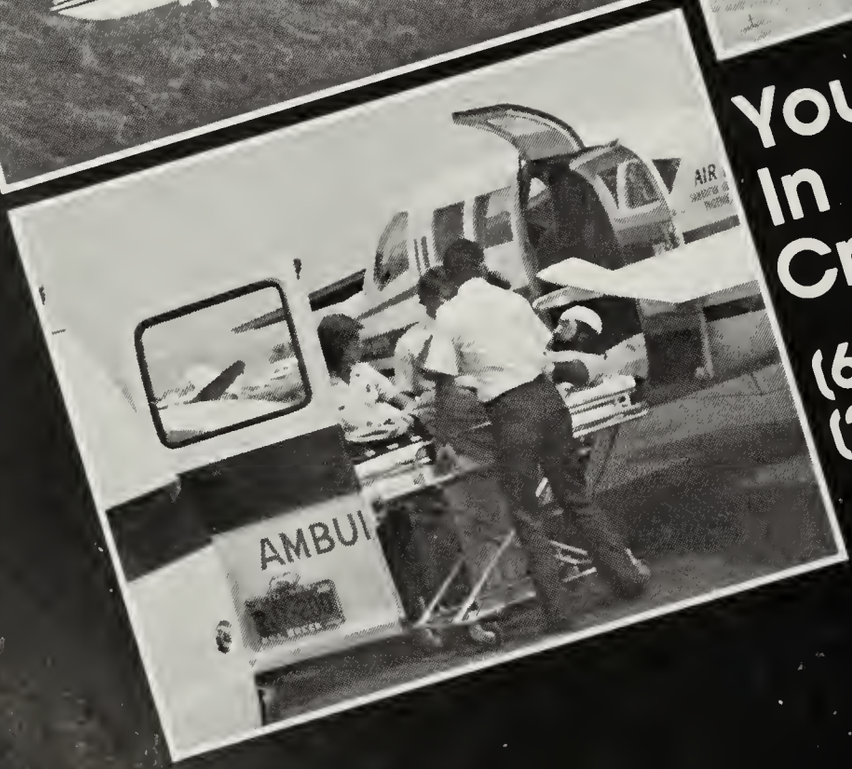
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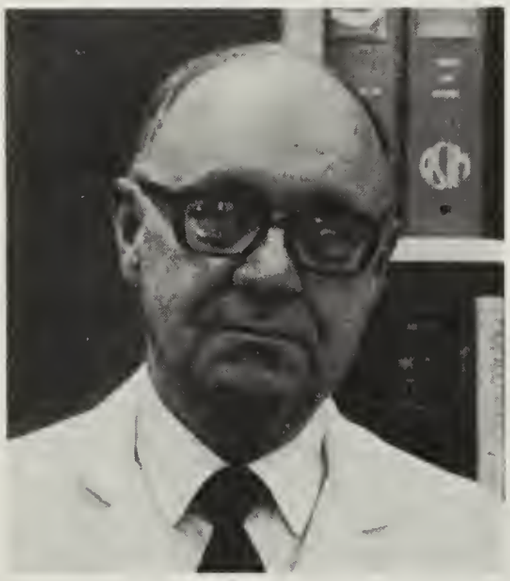


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More physicians today realize what a tough problem we're all faced with. They know this is a challenge for medicine. And that physicians are in the best position to deal with and solve the problem.

*PATIENT CARE Magazine—Outlook 1977, "Face-Off: Cost Containment vs. Chaos," January 1, 1977.

Lyle CB, et al. "Practice habits in a group of eight internists," ANNALS OF INTERNAL MEDICINE 84 (May 1976), 594-601.

Schroeder SA, et al. "Use of laboratory tests and pharmaceuticals: variation among physicians and effect of cost audit on subsequent use," JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION 225 (Aug. 20, 1973), 969-73.

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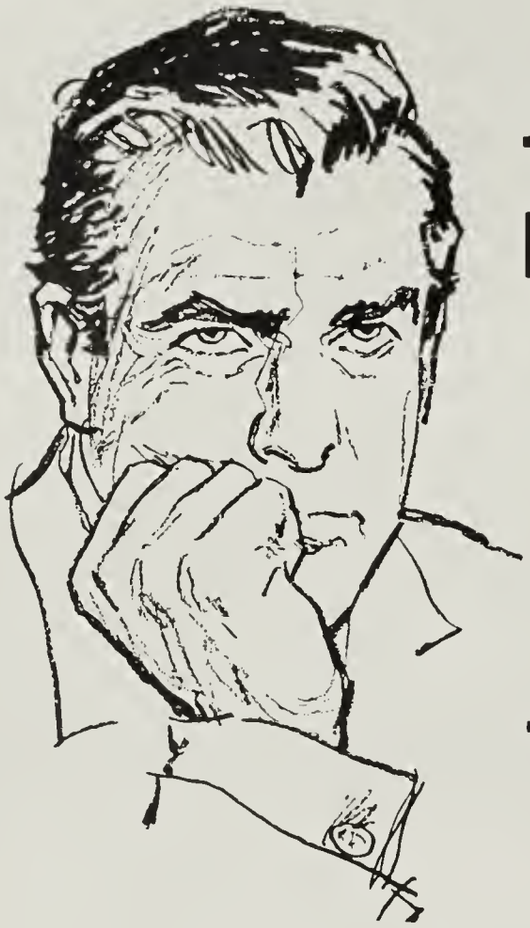
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Cardiology

Constrictive Pericarditis:
A Review

N. Sri Prakash, M.D.
Jeremy R. Torstveit, M.D.
Tong Ping Chee, M.D.
Alberto Benchimol, M.D.

Editor:
Kenneth B. Desser, M.D.

A description of constrictive pericarditis was first recorded by Chevers of Guy's Hospital in 1842. In 1896, Pick elaborated on the clinical and pathological features of this disease, which now bears his name.[1] Since these early reports the pathophysiological concepts and therapeutic modalities have been delineated and refined. This paper represents a review of the current literature on pericarditis citing the findings in patients we have recently evaluated and treated.

The Institute for Cardiovascular Diseases, Inc. and the Division of Cardiovascular Surgery, Good Samaritan Hospital, 1033 East McDowell Road, Phoenix, Arizona 85006. Supported in part by the E. Nichols and Kim Sigsworth Memorial Funds, and by the Institute for Cardiovascular Diseases, Inc. Reprint requests to Alberto Benchimol, M.D., Good Samaritan Hospital, Cardiovascular Institute, P.O. Box 2989, Phoenix, AZ 85029

Table 1
Etiological Classification of
Chronic Constrictive Pericarditis

1) Idiopathic
2) Specific Infections
 a) Bacterial Infections
 b) Tuberculosis
 c) Fungus Diseases
 d) Viral Disease, Coxsackie B-3 virus
3) Connective Tissue Diseases:
 Rheumatoid Disease
 Lupus Erythematosus
4) Neoplastic Diseases
 a) Primary: Mesothelioma or Sarcoma
 b) Secondary: Bronchogenic carcinoma, Breast Cancer, Lymphoma, Leukemia
5) Trauma, penetrating or non-penetrating
6) X-Irradiation
7) Uremia, especially after hemodialysis

Normal Pericardium

The normal pericardium forms a fibrous mantle around the heart, holding it in a fixed position in the thorax with 10-20 cc of fluid within lubricating the beating heart. It provides both physical protection and barrier to infection. The pericardium affects the systolic and diastolic functions of the heart.[2,3] It prevents acute dilatation of the heart chambers and under conditions of left ventricular failure, prevents pulmonary edema by limiting right ventricular filling and output.[4] Additionally, this structure facilitates right ventricular filling in normal subjects by the development of negative pressure in the pericardial sac during ventricular systole.

Constrictive Pericarditis

Constrictive pericarditis is the most common symptomatic variety of chronic pericarditis. A state of constriction is said to exist when there is impedence to diastolic filling of the heart. Ventricular contraction may also be hampered but this is not a necessary feature of the disease.

The causes of contrictive pericarditis are varied and an etiological classification of the disease is given in Table 1.[5] Before the antibiotic era a majority of cases of constrictive pericarditis were secondary to tuberculosis. Acute idiopathic pericarditis has been shown to provide a substrate for subsequent constriction. Constrictive pericarditis following open heart surgery has been attributed to hemopericardium, post-pericardiotomy syndrome, mediastinal plasminate infusion and to the practice of painting povidone iodine on the pericardium.[6] One of our patients had pericarditis secondary to long standing rheumatoid arthritis and a second subject developed the process as a result of therapeutic irradiation to the chest fourteen years prior to presentation.

Clinical Presentation

Patients usually present in the second to seventh decade of life. The most common initial symptom is dyspnea. Although the constriction of inflow to the right ventricle should allow less blood to flow through the pulmonary circulation, catheterization data have shown that pulmonary hypertension is present in most of afflicted patients. The other commonly encountered symptoms and signs are edema, abdominal swelling, palpitation and fatigue.

On examination such subjects appear healthy unless they present in late stages with orthopnea and cyanosis. The pulse is of small volume. Pulsus paradoxus is seen in approximately 15% of patients[7] and this sign was observed in both of our patients. Distention of the jugular veins is a hallmark of constrictive paricarditis.

Inspiratory augmentation (Kussmaul's sign) may be observed in a minority of patients. Careful observation of the jugular pulse reveals prominent "x" and "y" descents, the basis of which will be explained below.

Precordial palpation reveals a diminished cardiac impulse. The heart sounds may be distant. A pericardial "knock" is classically heard in patients with constrictive pericarditis. This distinctive finding is heard approximately 0.09-0.12 second after the aortic component of the second sound (Figure 1). Its occurrence is associated with an abrupt halt of rapid filling in early diastole owing to decreased ventricular compliance, small ventricular volume and high filling pressure. Murmurs are usually absent unless coexistent heart disease is present. The lungs are usually clear to ausultation. Hepatomegaly, ascites and peripheral edema are frequently observed in these patients.

Radiographically, the heart size may be normal or enlarged. Pericardial calcification is seen in 40 to 50 percent of cases. Fluoroscopy may reveal normal cardiac pulsations. The thickness of the pericardium can best be assessed angiocardiographically by examining the right atrial border which shows thickening, straightening and decreased motion in most cases. Ramsey et al[8] have shown that the left anterior descending artery is normally seen on the surface of the cardiac silhouette, but is placed deeper within, depending on the thickness of the pericardium, in patients with constrictive pericarditis.

Echocardiography has revolutionized the diagnosis of pericardial effusion and constrictive pericarditis. Figure 2 represents an example of pericardial thickening detected ultrasonically in a patient with radiation induced constriction. Careful attention to proper echographic technic is paramount since echoes from the region of the pericardium may provide a falsely thickened appearance.[9] Flattening of diastolic motion of the left ventricular posterior wall is a more characteristic sign, but this finding is also seen in restrictive cardiomyopathy and acute dilatation of left ventricle when the stiff pericardium exerts a constrictive effect. Other echocardiographic findings include paradoxical

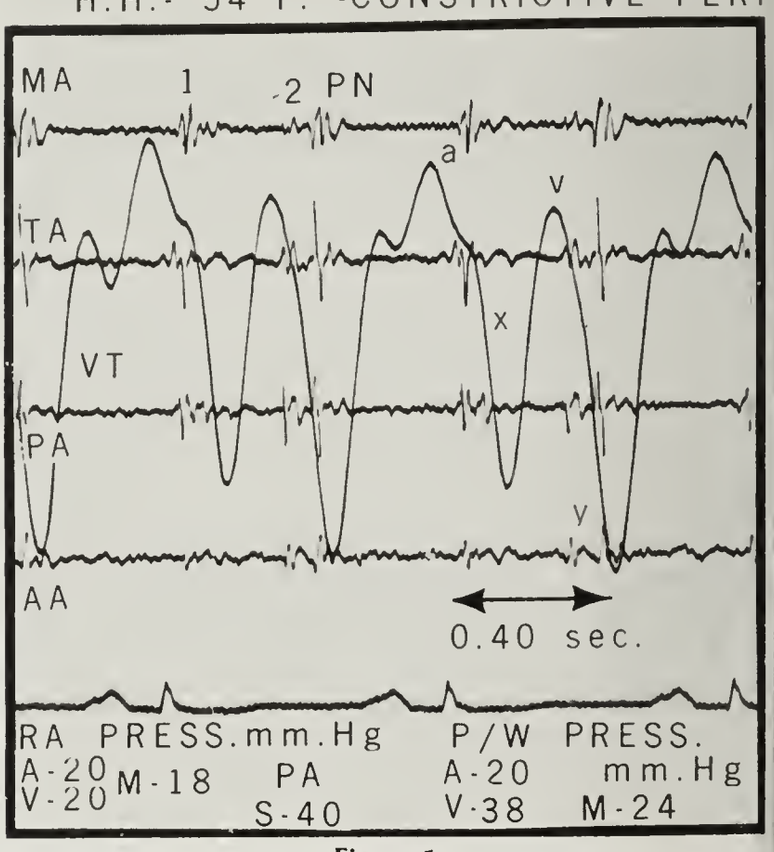


Figure 1

Simultaneously recorded mitral area (MA), tricuspid area (TA), pulmonic area (PA) and aortic area (AA) phonocardiograms, external jugular venous pulse tracing (VT) and lead II of electrocardiogram from a patient with constrictive pericarditis. Note the prominent pericardial knock (PN) which is recorded over the entire precordium. The VT shows prominent "x" and "y" descents. Right atrial (RA), systolic (s) pulmonary artery (PA) and pulmonary arterial wedge (P/W) pressures are indicated.

motion of the septum in systole, premature opening of the pulmonic valve and early closure of the mitral valve.[9]

Electrocardiographic changes are not specific. Diffuse ST-T changes such as T wave inversion or flattening and ST depression may be present. Atrial fibrillation is the most prevalent rhythm disturbance and is seen in approximately 25-35% of cases. Atrial flutter and paroxysmal atrial tachycardia have also been observed.[10] Abnormal Q waves are not uncommon and they may represent the presence of myocardial fibrosis and related to the pericardial process.[10]

Hemodynamic Studies

The most accurate method for the diagnosis of constrictive pericarditis is measurement of pressrue and flow in the heart. Before discussing the hemodynamic effects of constrictive pericarditis a brief presentation of normal blood flow and pressure is in order. The normal pressure in inferior and superior vena cavae exceeds atmospheric pressure by only a few millimeters of mercury. The jugular venous tracing (Figure 3) consists of positive a, c and v waves; "a" due to atrial systole, "c" due to the movement of the tricuspid valve during the isovolumetric phase of ventricular systole and "v" due to passive atrial filling. The negative wave "x" is caused by

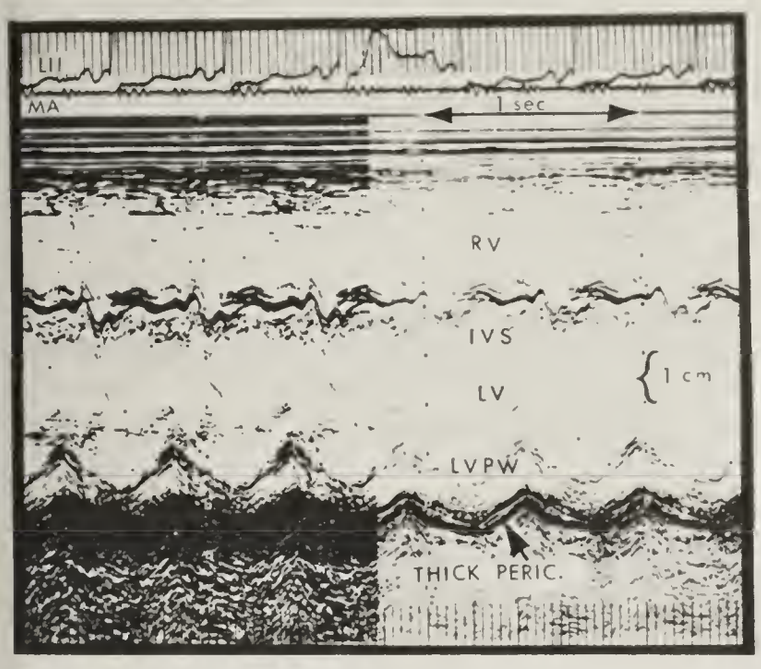


Figure 2
Echocardiogram from a patient with constrictive pericarditis secondary to mediastinal irradiation. Note the thickened pericardium (LII=lead II of electrocardiogram, MA=mitral area phonocardiogram, RV=right ventricle, IVS=interventricular septum, LV=left ventricle, LVPW=left ventricular posterior wall).

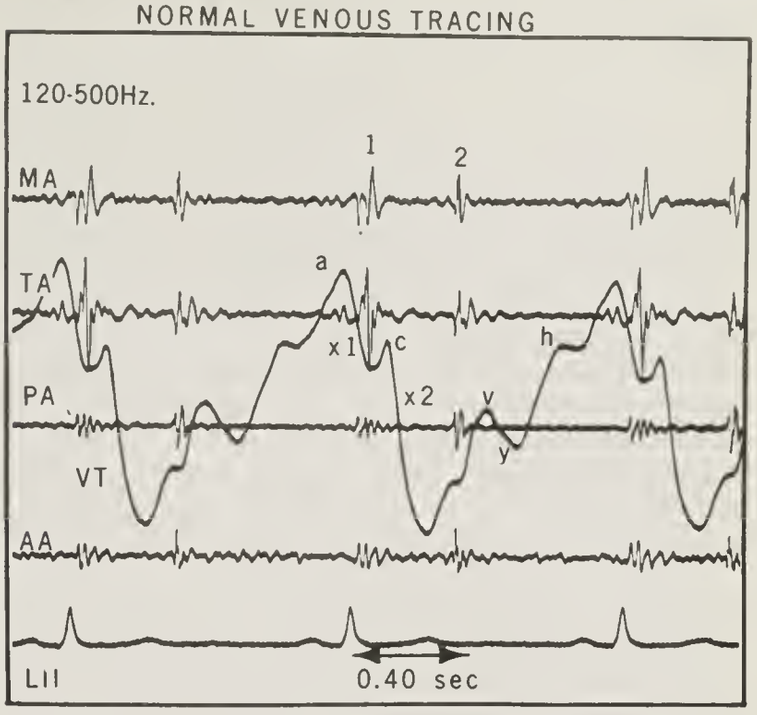


Figure 3
Phonocardiogram, jugular venous pulse tracing and electrocardiogram simultaneously recorded from a normal subject. Note the 2 components of the x descent (x1 and x2) interrupted by the c wave. The y descent is slow and shallow when compared with the x descent. (Abbreviations same as figure 1).

downward displacement of the tricuspid valve, but more so due to decreased intrapericardial pressure at the beginning of ventricular systole. This causes a sudden drop of pressure in the right atrium and blood flows from superior and inferior vena cavae to the right atrium. The "y" trough is caused by opening of the tricuspid valve and right ventricular filling. This also causes right atrial filling from the vena caval (Figure 4). The blood flow into the right atrium is biphasic in normal man and the pressure and flow have an inverse relationship. Since inspiration causes more negative pressure in the intrathoracic structures, the flow increases to the right side of the heart accounting for increased right atrial filling during that phase of respiration.

In constrictive pericarditis the mean venous pressure is elevated in direct relation to the degree of constriction. A characteristic "M" pattern of the right atrial pulse, early diastolic dip in both right and left ventricles along with elevation and equalization of the end-diastolic pressures of all the chambers of the heart are hemodynamic hallmarks of constrictive pericarditis (Figure 5). Hemodynamic data obtained in two of our patients are shown in Table 2. The deep "x" trough of jugular venous tracing corresponds to the beginning of right ventricular ejection and "y" trough to the rapid early diastolic dip. A late diastolic plateau of right ventricular filling causes marked elevation of the venous pressure. Right atrial filling is biphasic and corresponds to deep x and y troughs, but the respiratory variation of the flow is abolished. In occasional patients with severe

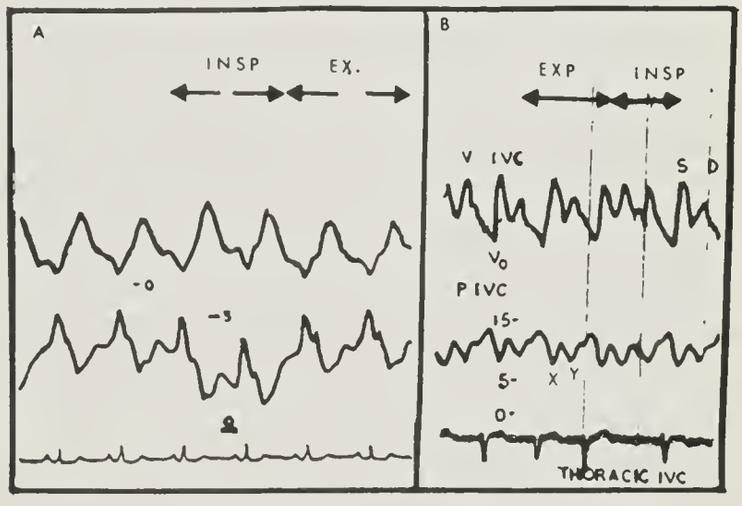


Figure 4
Simultaneous recording of respiration, caval blood flow, caval pressure and electrocardiogram from a normal subject (panel A) and a patient with constrictive pericarditis (panel B). Note that caval blood flow is enhanced at the same time that caval pressure falls during inspiration (INSP). The reverse effect is seen during expiration (EXP). Constrictive pericarditis influences hemodynamics such that there is no significant alteration of caval pressure or flow during the respiratory cycle. (From: Shabetai Reral: The Hemodynamics of Cardiac Tamponade and Constrictive Pericarditis. *AM J Cardiol* 26:480, 1970, with permission).

Table 2

	Case 1	Case 2
Cause	Rheumatoid Arthritis	Irradiation
Age	45 years	23 years
Sex	Male	Female
Pulsus Paradoxus	30 mmHg	12 mmHg
Right Atrial Pr. V-A-M	33-34-30	45-27-30
Right Ventricular Pr. Systole/Diastole	54/32	61/28
Pulmonary Artery Pr. Systole/Diastole	53/35	58/31
Pulmonary Capillary Wedge V-A-M mmHg	36-38-33	45-25-30

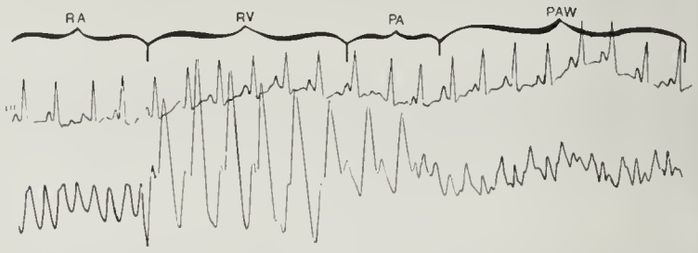


Figure 5
Right atrial (RA), right ventricular (RV), pulmonary artery (PA) and pulmonary arterial wedge (PAW) pressure curves from a patient with constrictive pericarditis secondary to rheumatoid arthritis. Characteristic findings are an "M" shape right atrial presssure contour, a marked diastolic dip of the right ventricular pressure and equilibration of right atrial and left atrial pressures.

constriction, inspiration is associated with an increase in vena caval pressure due to decreased right atrial filling.

Patients with cardiac tamponade frequently manifest similar pressure characteristics with a few important differences. The vena caval flow is monophasic occurring during "x" trough, the "y" descent is obliterated and at times transformed into an abnormal positive wave. The inspiratory augmentation of vena caval flow is preserved in cardiac tamponade. With obliteration of the "y" descent in the venous pressure the early diastolic dip of right ventricular pressure is absent in cardiac tamponade.[11]

The pattern of pressure plateau observed in constrictive pericarditis is also seen in patients with restrictive cardiomyopathy and myocardial fibrosis. The differentiation between these entities may only be apparent at thoracotomy on direct inspection of the paricardium. Although the hemodynamic pattern of cardiomyopathy occasionally resembles that associated with constrictive pericarditis, there are a few important differences; 1) higher left-sided pressures, 2) absence of early diastolic dip and plateau and 3) higher pulmonary artery pressure (usually >60 mmHg) are recorded in cardiomyopathy. A discussion of the differentiation between these entities can be found in an excellent paper by Meany et al.[12]

Bush et al[13] have described an intriguing variation which they called Occult Constrictive Pericardial Disease. These patients presented with vague chest pain, fatigue and dyspnea. On catheterization, patients were found to have normal hemodynamics at rest, but after rapid infusion of 1000 cc of normal saline, they developed marked elevation of central venous pressure. Identification of constrictive pericarditis depends on the appearance of typical pressure morphology from the right atrium, loss of respiratory variation and diastolic equalization of right and left heart pressures. Most of these patients underwent surgery and were found to have significant pericardial disease.

Hancock[14] has described an intermediary syndrome between chronic constrictive pericarditis and pericardial tamponade designated "Subacute Effusive Constrictive Pericarditis." Affected subjects with pericardial effusion did not have normal hemodynamics after reduction of intrapericardial pressure by pericardiocentesis. Before tapping they displayed a right atrial pressure contour comprised of predominant x or equal x and y descents. After the tap, their pressure configurations changed to a more prominent y descent, compatible with a constrictive picture. At operation these patients had thickened parietal and visceral layers with significant pericardial effusion. Some of their subjects were observed to progress through an effusive constrictive phase to non-effusive constrictive pericarditis, pericardiectomy was curative in all these patients.

Treatment

The therapy of choice for constrictive pericarditis is pericardiectomy. However, many of these patients can be kept asymptomatic with vigorous diuretic therapy. It is a matter of judgment regarding the timing of operation. Each subject must be evaluated individually for incapacitating symptoms and treatment decided. In recent years the mortality associated with surgery has dropped considerably. Wychulis et al[15] reported an operative mortality of 6 percent compared to previous figures of 11-24 percent.

The improvement of hemodynamics following pericardiectomy is not immediate. Viola[16] followed six patients who underwent pericardiectomy for constrictive pericarditis of varying duration, by repeated right heart catheterization. No significant changes were observed immediately following surgery. The right-sided pressures showed a downward trend by the second to fourth day. Two to six months after surgery all these patients were found to have normal hemodynamics. During the immediate postoperative period these patients may require aggressive fluid administration despite elevated pressures. Medical treatment with diuretics may have to be continued until their hemodynamic state normalizes. Our patient with constrictive pericarditis secondary to rheumatoid arthritis showed persistent elevation of right-sided

pressure and cardiac dilatation one week postoperatively. At the follow-up examination four months later, he had no abnormal venous distention, diminution of the heart size radiologically and required only small doses of diuretics.

The major causes of surgical failure are: 1) Incomplete decortication of the ventricles and the atrio-ventricular groove and 2) Myocardial fibrosis. Myocardial fibrosis may represent an extension of the inflammatory process from pericardium to myocardium or arise secondary to partial ventricular immobilization from long standing constriction. These considerations suggest that earlier surgical intervention be entertained in patients with constrictive pericarditis who do not have major symptoms.

Acknowledgement

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Editorial Comment

Kenneth B. Desser, M.D.

Prior to 1960, a few cases of constrictive pericarditis were usually encountered each year in teaching hospitals. During the next decade the frequency of such patients diminished to the point where they became Grand Rounds Cases. It has been inferred that the diminished prevalence of the disease was related to improved control of infectious processes and specifically tuberculosis. The incidence of constrictive pericarditis appears to be increasing and the most commonly recognized causes are not infectious. Of special interest are those cases where the constrictive effect arises as a consequence of therapeutic interventions. Examples include: 1) *radiation therapy* where cure of mediastinal lymphoma is followed by a particularly aggressive pericarditis which can smolder for years; 2) *aortocoronary bypass* where the process can result from pericardial incision or manipulation, utilization of irritating antiseptics, mediastinal hemorrhage or infusion of plasma expanders and 3) *epicardial pacemaker insertion* where hemorrhage from the site of myocardial electrode implantation provides the substrate for inflammation, organization and fibrosis.

These etiologies are being reported with increasing frequency. Unfortunately, these causes and others such as connective tissue disorders (eg. rheumatoid arthritis) pose a specific therapeutic problem. In contrast to "idiopathic" or tuberculous pericarditis there is usually a major myocardial component in association with the constrictive process and often pericardial resection results in minimal improvement of symptoms. Despite major advances in hemodynamic, angiographic and radionuclide assessment of myocardial function it is impossible to predict the degree of improvement following surgery in the individual case.

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Seminars in Dermatology

Porphyria Cutanea Tarda

Robert L. Anderton, M.D.

Editors:
Robert A. Schwartz, M.D.
Ronald C. Hansen, M.D.

Porphyria cutanea tarda (PCT) is an abnormal metabolic process presenting as increased skin fragility, associated with scarring, vesiculation, and milia production. Frequently, hypertrichosis over the face, as well as sclerodermoid tightening of the skin may be present.

This disease is noted in mid to older age and invariably is precipitated by liver disease. The hepatotoxins have included the insecticide hexachlorobenzene (1937)[1] as well as estrogenic hormones,[2] but by far the commonest hepatotoxin still is ethanol.

Harris first recognized the disorder in 1898,[3] but Waldenstrom (1927) described the symptomatic and hereditary type. Kushner (1976)[4] determined that the disease was predictably associated with a decrease in both hepatic and erythrocyte uroporphyrinogen decarboxylase activity. He was able to demonstrate this enzymatic inherited disorder in unaffected relatives of those clinically affected persons. Six cases have been diagnosed in a rural Arizona community (Table 1). The following case report is typical.

A 64-year-old white female presented with a blistering disorder on the dorsum of her hands. She consumed approximately 2 ounces of bourbon nightly and had been on estrogen for menopausal symptoms. Physical exam revealed facial hypertrichosis as well as milia, erosions and vesicles over the dorsal hands. No sclerodermoid thickening was noted. Lab work revealed normal fasting glucose, Fe, FeBc, Hbg/Hct. Liver biopsy indicated fatty metamorphoses with increased liver iron stores. No cirrhosis was evident. Skin biopsy revealed a subepidermal blister with PAS + hyaline amorphous deposition around vessels consistent with PCT. Urinary studies showed respective quantitative values for uro and coproporphyrins of 2800/520 mcg per 24 hours (Table 2, Case 2).

The diagnosis of PCT is confirmed by quantitative 24 hour urine assay for uro and coproporphyrins. Invariably the uro is elevated more than the coproporphyrins (see Table 2). Associated features may include increased body iron, and a tendency toward diabetes. Skin biopsy from affected vesicles/bullae reveal a subepidermal blister with a corrugate base and endothelial small vessel proliferation. A PAS + amorphous material is deposited proximate to the vessels. Liver biopsy reveals increased iron stores and fatty changes, but generally not far advanced cirrhosis.

From: 2451 South Avenue A, Yuma, Arizona 85364.

Table 1

Case Per Table Two	PT	Age	Duration In Years	Features	Glucose Fe, FeBc	Skin BX	Liver BX	Drugs HX
6	L.A.	60	4	erosions, papules, onychodystrophy, sclerodermoid scalp, milia	127 191/222	NA	NA	Ethanol++ 3-5 beers/day x 20 years
4	D.F.	45	2	hypertrichosis erosions milia, hemorrhagic vesicles, hyperpigmentation	100 57/208	porphyria, festooning of dermal papillae perivascular PAS + deposition	fatty liver ↑Fe stores	2 qts/wk for years
5	C.D.	48	8-10	erosions, ulcers, milia	105 143/298	NA	NA	6 beers/day
1	S.N.	38	3	Hypertrichosis, hyperpigmentation, vesicles, erosions, milia	NA	NA	NA	heroin addiction, alcohol excess
3	N.D.	59	6	vesicles, milia	NA	NA	NA	Premarin, Fe supplement
2	B.B.	64	4	hemorrhagic vesicles, milia, hypertrichosis, scars	87 153/279	subepidermal bilster with festooning dermal papillae + thickened vessel walls	fatty metamorphosis + ↑Fe stores	3 oz/day burbon x 5 years, estrogen

The signs of fragility and bullae appear on sun exposed skin and are related to a photoreaction dependent on UVA (400 nM) light. The porphyrin ring is chemically excited thus leading to oxidized by-products (peroxides) which damage the vessels and lead to vesicle formation, scarring and milia. The etiology of hypertrichosis is unknown, but androgenic stimuli has not been shown.

Treatment always is initiated by elimination of the hepatotoxin. Weekly phlebotomy[5] done as tolerated, leads to decreased uroporphyrin levels and eventually 6 to 9 months later to clinical improvement as well (Table 2, Case #2,4,5,6). Another controversial form of therapy is alkalinization therapy with $NaHCO_3$.[7] Most recently antimalarial (Chloroquine)[8] therapy results in a flushing or purging. All forms of treatment must be instituted only after the hepatotoxin has been eliminated.

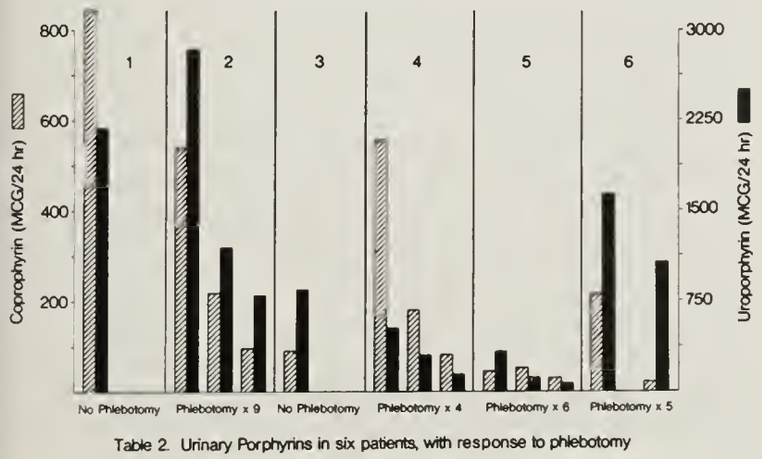


Table 2. Urinary Porphyrins in six patients, with response to phlebotomy

As we can see from the charts, six patients have been presented from a rural Arizona community with both chemical laboratory and clinical confirmation of this unique dermatologic disorder. We Arizona physicians should reacquaint ourselves with PCT as the intense sun exposure of our patient population makes it a very obvious clinical problem.

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Seminars in Gastroenterology and Liver Disease

Diarrhea or Dysentery?

Pathogenesis, Diagnosis and Therapy

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Editors:
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In the past, we have tended to use the term "diarrhea" loosely. We have thrown patients with one watery stool a day into the same hopper with those who have three mushy stools per day and labelled them all "diarrhea." With our expanding knowledge of the pathology and normal physiology of the gastrointestinal tract, we have been able to draw important distinctions among patients with abnormal stool patterns—distinctions invaluable in helping us to understand, diagnose and treat these individuals.

At the outset, it is necessary to make an important distinction between diarrhea and dysentery. Diarrhea is the malabsorption of water and electrolytes.[1] The pathology productive of this state almost always lies in the small intestine. Under normal circumstances, there is a bidirectional flux of sodium and water within the small intestine. Sodium and water are simultaneously absorbed by the cells lining the villi and secreted into the lumen by the cells lining the crypts. When we say that the small intestine absorbs sodium and water, we really mean that the net effect of the bidirectional movement of sodium and water favors absorption. Thus, diarrhea may be produced by a process that impairs function of the absorptive cells on the villus surface; this may occur, for example, in viral enteritis, giardiasis or in celiac disease. Alteration of normal mucosal transport may also be secondary to vastly increased function of small intestinal crypt cells resulting in excessive secretion of water and electrolytes into the bowel lumen; this state is classically produced by bacterial toxins but may also be produced by celiac disease or circulating peptide hormones. Diarrhea may also be produced by increase osmolality of luminal contents; this may be secondary to disaccharidase deficiency which in turn may be produced by bowel infection, malnutrition or again, even by celiac disease. Under these circumstances, an unabsorbed disaccharide may travel down the bowel lumen dragging an osmotic equivalent of water with it. Finally, diarrhea may even be secondary to increased bowel motility. This possibility is suspected because of the accumulation of hormones such as prostaglandins and vasoactive intestinal peptide (VIP) within the bowel in certain infectious states; these hormones increase motility of intestinal smooth muscle.[2]

On the other hand, dysentery refers to loose stools containing blood and inflammatory detritus such as fibrin and polymorphonuclear leucocytes. Dysentery is secondary to colonic mucosal inflammation. Such inflammation may be produced by invasive bacteria such as Shigella, Salmonella or Campylobacter but may also be produced by parasites such Entameba histolytica, by bacterial toxins such as that found in antibiotic-associated dysentery and by ulcerative or granulomatous colitis.

Patients with diarrhea and dysentery differ with respect to history and a few simple and well placed laboratory tests. However, some patients such as those with Campylobacter enteritis may present clinical features of both while other patients with Shigella or Salmonella enteritis may first present with diarrhea and only later develop the more classical dysentery. Patients with typical diarrhea (i.e. - patients with small bowel fluid and electrolyte malabsorption) have watery stools. They have little or no fever and usually deny abdominal cramps. Their stools do not contain either blood or polymorphonuclear leucocytes and they do not have leucocytosis, a shift to the left or an elevated sedimentation rate. Patients with classic dysentery (i.e. - patients with colonic inflammation) have mushy stools. They frequently have fever and prominent abdominal cramps that are associated with and relieved by defecation. Their stools often contain blood and polymorphonuclear leucocytes. They also have leucocytosis, a shift to the left and an elevated sedimentation rate.[3]

At this point, we will examine in greater detail some of the entities responsible for diarrhea and dysentery. Many infectious agents including viruses, parasites and a variety of bacteria are responsible for the small intestinal effects productive of diarrhea. Only a handful of viral agents causing diarrhea have been well studied.[4,5] Among these are the Norwalk and Hawaii agents and human rotavirus-like agent. None of these viruses has ever been shown to activate intestinal cyclic-

From: Tucson Hospitals Medical Education Program, Tucson Medical Center, Tucson, Arizona. Reprint requests to Dr. Stillman at 5200 E. Grant Road, Ste 611, Tucson, AZ 85712.

AMP. All of them produce diarrhea by limiting the absorptive capability of the small bowel and consequently clinical diarrhea is manifest primarily in patients who eat and tends to decrease in patients who are fasting. Vomiting and mild fever are frequently associated with viral diarrhea. The viruses infect only the small intestine and morphological recovery occurs within several weeks of infection corresponding to the rapid clinical recovery. Radioimmunoassay detection of virus in stool and serum antibody titers against specific viruses are available. However, both the viruses and the diarrheas they cause are usually transient and such diagnostic tests are often an academic exercise.

Giardia lamblia is an intestinal protozoan that lives high in the small intestine.[6] Despite intensive study, the pathogenesis of diarrhea caused by this organism is unknown. The parasite causes transient intestinal lactase deficiency which may contribute to the diarrhea. Giardia has recently been shown to deconjugate bile salts; this may be at least partially responsible for the fat malabsorption associated with many cases of infestation with this parasite.[7] Giardia also causes partial or rarely, total villous atrophy, thus limiting intestinal absorptive capability. This parasite causes chronic continuous or intermittent diarrhea and may persist for years if untreated. Only one-third to one-half of cases of giardiasis are found by stool examiantion; however, up to ninety percent of cases are detectable by examination of upper small bowel contents either by intubation or by collection of bowel mucus with a weighted capsule which the patient swallows. The capsule carries with it a string that resides within the small bowel for several hours and is then withdrawn so that mucus collected on the string can be microscopically examined for the parasite. This test, called an Enterotest, is available at any GI laboratory.

Various bacteria produce diarrhea through production of toxins that activate cyclic-AMP or cyclic-GMP and thereby vastly increase the secretory function of the crypts.[2] Classically, Vibrio cholera and E. coli produce such toxins. Even typically invasive organisms such as Shigella and Salmonella have been thought to produce toxins affecting regions of the small intestine distant from actual bacterial invasion. In a recent article, an additional mechanism of bacterially mediated diarrhea was proposed in which a nontoxigenic, non-invasive strain of E. coli produced diarrhea by adhering to the mucosal surface and possibly by destroying the microvilli on the surface of the absorptive cells.[8] (Microvilli are corrugations of the cell membrane on the luminal surface of the absorptive cell that vastly increase the absorptive surface of each cell).

Increased luminal osmolality may be secondary not only to disaccharidase deficiency, whether primary or secondary, but may also be due to surreptitious ingestion of laxatives such as milk of magnesia. This laxative depends for its effects on the limited ability of the small intestine to absorb magnesium. Following ingestion, a large quantity of unabsorbed magnesium travels down the length of the small intestine dragging with it an osmotic equivalent of water which is unable to be adequately absorbed in sufficient time by the colon.

Finally, diarrhea may be secondary to hormonal mediation. The carcinoid syndrome, for example, is associated with high levels of serotonin which may increase bowel motility. Agents such as prostaglandins and VIP have been found within the bowel lumen in infectious disease states such as cholera. These agents contract intestinal smooth muscle thus increasing bowel motility and have also been thought to activate cyclic-AMP thereby increasing salt and water secretion into the small intestinal lumen.[2]

The following section reviews conditions productive of dysentery. Here again, infectious agents are especially prominent causes. Shigella is an invasive organism that is capable of causing severe dysentery. In the case of Salmonella, all forms of the organism causing clinical disease invade the distal ileal mucosa; in addition, some forms invade the colonic mucosa and cause typical ulceration and dysentery. Patients with Shigella and Salmonella colitis present with abdominal cramps, fever and loose stools that often contain gross blood. Shigella uncommonly gives positive blood cultures. In some strains of Salmonella, the incidence of positive blood cultures achieves twenty-five percent.

Campylobacter is a newly recognized invasive human pathogen that is now thought to be accountable for anywhere from four to fourteen percent of enteric infections.[9] The transmission of the organism may be either person-to-person or from a common source, frequently an animal (such as a dog) or through contaminated milk. The disease usually begins with severe abdominal pain and fever. Profuse watery diarrhea may occur either at the onset or follow the appearance of abdominal pain within one to three days. Bloody stools occur frequently. The diarrhea usually lasts three to five days and ninety percent of affected individuals are asymptomatic within two weeks of the start of illness. Death due to Campylobacter enteritis is rare. Diagnosis of the organism is proved with difficulty; Campylobacter grows on antibiotic-impregnated media at low oxygen tension. If Campylobacter is suspected, the laboratory should be altered to the necessity for special culture media.

The clinical features of amebiasis in our area are well known.[10] Amebiasis can produce any or all of the clinical features we associate with inflammatory bowel disease. The enteritis begins with watery or mushy bowel movements that may contain gross blood but almost always contain occult blood. The patients often have abdominal pain and leucocytosis. Examination of the stools fails to reveal evidence of polymorphonuclear leucocytes; Entameba histolytica and Salmonella typhosa are the two notable examples of potential dysenteric organisms that do not produce a stool poly response. Diagnosis is achieved by examination of warm

stool smears from individuals who have not had preceding barium enemas or other interfering substances such a mineral oil, castor oil or antibiotics. Under these circumstances, a skilled parasitologist can make the diagnosis on three fresh stools with a confidence of greater than ninety percent. Amebic serology by indirect hemagglutination test is positive in seventy-five percent of dysenteric cases.

Dysentery maybe produced by inflammatory bowel disease, namely ulcerative colitis and Crohn's disease of the colon. These conditions are diagnosed by history, sigmoidoscopic findings and typical x-ray findings. They are amenable to therapy with either steroids, salicylazosulfapyridine or a combination of these; ultimately, surgery may be necessary for control.

Finally, antibiotic-associated colitis is now known to be secondary to overgrowth of the bowel by Clostridium difficile in response to a change in colonic flora induced by a wide variety of antibiotics.[11] This organism produces a toxin that causes severe morphological damage to the colonic mucosa. A large number of antibiotics have now been associated with this syndrome which is typically ushered in by watery diarrhea and whose classical and virtually pathognomic sign is the typical sigmoidoscopic appearance of adherent white purulent plaque that is wiped off the mucosa only with difficulty and produces hemorrhage at the site of removal. The condition is suspected by clinical history and sigmoidoscopic findings just described may not be present within the first 25 cm or recto-sigmoid but may only be present in more proximal areas of the colon and are therefore diagnosed only by colonoscopy.[12]

Investigation of patients with diarrhea or dysentery is a logical process whose direction is suggested by the initial history and laboratory findings. For patients with history and laboratory findings suggestive of diarrhea, one can initiate investigation with stool cultures and examinations for ova and parasites. Cultures may pick up the case of toxin-producing Shigella or Salmonella presenting with diarrhea while parasite examinations may disclose the occasional case of giardiasis. If these examinations are unrewarding, the patients may be placed on an NPO regimen; cessation of the diarrhea when the patient is fasting immediately incriminates an osmotically active substance as the cause. This can easily be confirmed if, when the patient is eating his usual diet, the osmolality of a fresh stool specimen is 100 or more mosm greater than twice the sum of the stool sodium and potassium concentrations. (Under normal circumstances, the osmolality of fresh stool is almost completely accounted for by its electrolyte content). If an osmotic diarrhea is found, a lactose tolerance test should be performed and if positive, especially in a Caucasian subject, a small bowel biopsy should be considered to investigate the possiblity of lactase deficiency secondary to a primary small bowel condition such as celiac disease. If an osmotic diarrhea is not present, an Enterotest for giardiasis should be obtained. A small bowel barium x-ray should be performed in chronic cases to investigate the presence of intestinal stasis syndromes such as multiple jejunal diverticula, scleroderma, surgical blind loops, etc. If stasis exists, small bowel cultures may be obtained through intubation. The proximal jejunum should ordinarily contain fewer than 10^4 bacteria per cc intestinal contents and no coliforms or obligate anaerobes should be present.[13] If these studies are unrewarding and if the patient's clinical history is appropriate, he may be investigated for a hormonally responsive diarrhea such as carcinoid syndrome, Zollinger-Ellison syndrome, ganglioneuroma, adrenal insufficiency, diabetes mellitus or even VIP-induced diarrhea; the latter condition, while rare, is associated with massive diarrhea, hypokalemia, hyperglycemia and hypercalcemia.[14] If these measures do not produce an answer to the patient's problem, the possibility of surreptitious laxative abuse must be considered; the patient's stool may be submitted to the laboratory for analysis for phenolphthalein, the active ingredient in the most common and easily obtainable over-the-counter laxatives. If this analysis is negative, the patient may be sent from his room on a pretext and his room searched for laxatives.

On the other hand, if the patient presents with history and laboratory studies more consistent with dysentery, the patient's stool should also be submitted for culture and for parasite examination. If these are unremarkable, the patient should have a sigmoidoscopic examination. If an abnormal sigmoidoscopic picture is encountered, biopsies should not be obtained but the patient should be sent for barium enema to determine the extent of the inflammatory process; a rectal biopsy taken at this time would prevent safe performance of a barium enema for approximately two weeks. A normal sigmoidoscopic examination would also naturally prompt a barium enema as the next step. Following barium enema, signoidoscopic examination may always be repeated and the necessary biopsy obtained at that time. Colonoscopic examination is uncommonly indicated in the evaluation of dysentery. However, colonoscopy may be necessary in the event of a barium enema examination yielding equivocal results in the face of stools negative for culture and parasites.

The most important consideration influencing the therapy of diarrhea is that the great majority of attacks are transient and short. It is therefore most important to preserve the patient's nutrition and fluid and electrolyte balance until gastrointestinal physiology returns to normal. Following recovery, it may be necessary to avoid lactose for weeks to months until lactase content of the bowel is restored. If lactase deficiencey is responsible for a patient's diarrhea and his small bowel biopsy is normal, therapy involves only provision of a lactose-free diet. If small bowel bacterial overgrowth is discovered and an anatomical defect such as obstruction or a blind loop is found, corrective surgery is indicated. If a medical type

of stasis syndrome such as scleroderma is discovered, the patient can be treated with multiple courses of antibiotics such as tetracycline to eliminate the bacterial overgrowth. Acute bacterial diarrheas affecting the small intestine generally clear spontaneously within days to weeks of onset; antibiotic therapy generally offers little advantage in shortening the patient's course. Giardiasis is treatable with quinacrine, furazolidone, or with metronidazole. Metronidazole is not officially approved for use in giardiasis by the Food and Drug Administration; although it is therapeutically highly successful, the drug has been associated with mutations in bacteria and birth defects in animals. Celiac disease requires institution of a gluten-free diet. Clincial recovery occurs within weeks to months although the small bowel biopsy may remain abnormal for years after strict adherence to such a diet. Diarrhea due to carcinoid syndrome can be treated by institution of serotonin antagonists or cytotoxic agents.[15] VIP-mediated diarrhea is treated by angiographic localization of the hormone-producing tumor and its surgical excision, when feasible, and by streptozotocin in metastatic cases.[16] Zollinger-Ellison syndrome may be most effectively treated with the least number of complications by a combination of surgical vagotomy and cimetidine therapy.[17] Diarrhea due to ganglioneuroma is amenable to localization and surgical excision of the tumor. Diarrhea due to adrenal insufficiency rapidly responds to steroid replacement. Diabetic diarrhea, which is usually due to autonomic neuropathy, is often unresponsive even to strict blood glucose control although it may respond to loperamide. Patients who abuse laxatives require psychiatric evaluation.

The therapy of dysentery varies with the infectious agent. All cases of Shigella enteritis should be treated. In the United States, Shigella has become sophisticated and is now resistant to ampicillin in many areas of the country. Depending upon local antibiotic sensitivities, the drugs of choice are either ampicillin or trimethoprim-sulfamethoxazole. We know that most cases of Salmonella enteritis clear spontaneously; treatment of the routine case of salmonellosis only prolongs the carrier state and results in antibiotic-resistant strains and symptomatic relapses.[18] Therapy of Salmonella should therefore be limited to cases of unusual severity or those associated with septicemia. Recent literature indicates that Campylobacter may be sensitive to erythromycin.[9] Antibiotic-associated dysentery is amenable to therapy with oral vancomycin; in the absence of vancomycin, erythromycin is an effective substitute[11] and a recent publication states that bacitracin may also be similarly effective.[19] Amebiasis is easily treated with metronidazole. Inflammatory bowel disease is treatable by salicylazosulfapyridine, steroids or even by surgery.

In closing, a discussion of certain non-specific anti-diarrheal agents is appropriate. So-called anti-motility drugs such as codeine, opium, diphenoxylate and loperamide reduce abdominal cramps and stool frequency in many cases. However, although they may relieve symptoms, they may allow accumulation of intestinal secretions within the lumen of an immobile gut thereby predisposing to unsuspected dehydration. Moreover, by decreasing bowel motility they may prevent normal cleansing of the bowel, an action that may be especially injurious in Shigella and Clostridial colitis. Reduction of bowel motility in inflammatory bowel disease may predispose to toxic megacolon. A recent article failed to show an objective effect of diphenoxylate-atropine (marketed as Lomotil) compared to placebo in decreasing the number, weight or liquidity of stools of a group of patients with non-specific diarrhea.[20] Ingestion of bismuth subsalicylate as Pepto-bismol may decrease stool frequency in toxigenic E. coli-induced travellers' diarrhea but the volume of bismuth required (60 cc ever 30 minutes for eight doses) is itself a drawback to treatment.[21] However, another article indicated the potential usefulness of bismuth subsalicylate in decreasing severity and duration of symptoms associated with viral diarrhea.[22] Because viruses do not either produce a known toxin or activate cyclic-AMP, these authors suggest that bismuth may have a non-specific effect of decreasing gut neuromuscular irritability.

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Neurology

Temporal Arteritis

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Temporal arteritis is a common clinical disorder that frequently poses a difficult diagnostic problem. A review of the subject follows two case reports of unusual presentions.

Patient 1

A sixty-four year old white male presented with an eight-year history of bilateral arm and shoulder pain associated with digital pallor and acceleration of symptoms prior to admission. He also complained of six months of malaise, intermittent diplopia, and decreased exercise tolerance. His past medical history was significant for atherosclerotic heart disease with a myocardial infarction 16 years ago, chronic obstructive lung disease, and polycythemia associated with high altitude hypoxemia. Physical examination was remarkable for absent radial and ulnar pulses bilaterally, with good carotid pulses and the absence of neck bruits. Laboratory abnormalities included a hemotocrit of 38.1% with borderline hypochromic/microcytic indices and a sedimentation rate of 53 mm. and 70 mm. An arch aortogram revealed bilateral axillary artery stenosis with complete obstruction on the right with collaterals; and 95% obstruction on the left with recanalization, moderate proximal stenosis of the left subclavian artery with post-stenotic dilatation and mild stenosis of the right subclavian artery. The patient underwent a left dacron carotid subclavian graft with a left saphenous vein axillobrachial bypass followed by a right axillobrachial artery bypass and saphenous vein graft. A 6 cm. segment of nonpulsatile thickened segment of right axillary artery was noted with cross sectional biopsy submitted for pathological review. Histology was consistent with giant cell arteritis. The patient had an uncomplicated post-operative course. Sedimentation rates returned to normal after two weeks of high dose steroids (60 mg. Prednisone per day).

Patient 2

A sixty-four year old white female was admitted in September of 1978 with an acute onset of severe diffuse headache associated with nausea and vomiting that was followed by focal seizure activity. Her past medical

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history was remarkable for a twenty-four-year history of bilateral .throbbing headaches associated with scintillating scotoma that was previously diagnosed as a migraine syndrome. She had an EEG one year prior to admission that demonstrated right hemisphere spike and wave activity. She also has a history of abdominal pain with numerous evaluations but negative workups; associated with this was an anemia of unknown etiology and an elevated sedimentation rate. A review of systems revealed an 18-month history of a 15-pound weight loss and profund malaise. The neurologic exam was normal except for disorientation to time and place, coarse nystagmus on lateral gaze and a resolving left hemiparesis. Laboratory data was notable for a hematocrit of 31% with normal indices, a sedimentation rate of 35 mm. (previously 50-75 mm.), a nonreactive ANA, and CT scan of the brain that revealed a degree of atrophy which was considered normal for age. The patient subsequently underwent a temporal artery biopsy that was consistent with giant cell arteritis. She was treated as an outpatient with steroids. It was noted that her headaches and abdominal pain worsened on attempts to lower her doses of steroids. In December of 1979 she presented with an acute abdomen after a sudden onset of severe epigastric and right upper quadrant pain. After surgery, she was found to have generalized peritonitis secondary to perforated ileum that was felt to be due to mesenteric arteritis with ischemia. After a small bowel resection, she subsequently was placed on high dose steroids again, with resolution of her prior headache and abdominal pain and reduction of her sedimentation rates to normal.

Discussion

Temporal arteritis (also known as giant cell arteritis or cranial arteritis) is a disorder of unknown etiology, found almost exclusively in the elderly population. It was previously thought to be a rare, localized arteritis. It is not uncommon and it is now know to be a widely disseminated arteritis capable of involving any large or medium-sized elastic artery.

Polymyalgia rheumatica is a disease which effects the same age group and has similarities that suggest a common pathogenesis. Although this article will deal primarily with temporal arteritis, the relationship between these two processes will be incidentally discussed.

Historically, polymyalgia rheumatica (PMR) was originally described by W. Bruce in 1888. He erroneously labeled this process "senile rheumatic gout." Two years later, Jonathan Hutchinson published the first description of temporal arteritis (TA). He described an elderly man who couldn't tolerate his hat because of inflammed and tender temporal arteries. In 1932, Horton and associates reported several cases of granulomatous arteritis of the temporal arteries and stressed the widespread systemic manifestations as well as the significance of headache and jaw claudication. It wasn't until 1951 that Pasman first called attention to the similarities between PMR and TA. Alestig and Bair in 1963 were the first group to study temporal arteritis in patients with PMR, but clinically normal vessels and without headache or tenderness. Arteritis was found on biopsy in seven of nine patients. This study was a major contribution to understanding the relationship between PMR and TA.

The incidence of TA ranges from 2.4 per 100,000 for the general population to 17.4 per 100,000 for those over age 50.[1] The maximal incidence occurs in individuals over age 70. TA is rarely seen in patients less than 50 years old, and the diagnosis should be made with caution in this age group.[2] Geographically, there is an increased incidence of TA in northern climates. The racial distribution is more distinctive: TA is very uncommon in Orientals, Blacks and American Indians.

The histology of TA may be classified into three general patterns: (1) The classic presentation of granulomatous arteritis with Langhans giant cells and with the major involvement occuring in the media of the artery with smooth muscle and internal elastic membrane necrosis; (2) a nonspecific arteritis with an inflammatory reaction involving the entire arterial wall with an exudate of lymphocytes, eosinophils, plasma cells and less frequently, neutrophils, and (3) sclerosis with intimal thickening and calcification in the media with occlusion of the vessel lumen.[3] It is felt that these patterns may reflect the natural course of the disease rather than different modes of pathologic presentation.[4] There may be "skip lesions" which are isolated areas of normal histology along the course of an artery with active arteritis. The clinical severity does not correlate with the presence of skip lesions versus continuous arteritis. Also, there is no correlation between the histopathologic appearance of the biopsy and duration of symptoms. It is of interest that morphologically, TA is indistinguishable from Takayasu's arteritis (pulseless disease), which, in contrast to TA, affects a younger population, occurs more frequently in non-Caucasians and is relatively resistent to corticosteroid therapy.

The most common sites of involvement include the ophthalmic and posterior ciliary branches of the internal carotid system; the superficial temporal, maxillary, facial and occipital branches of the external carotid system; vertebral arteries, aortic arch, abdominal, mesenteric and other large and medium-size arteries. The absence of involvement of the renal vasculature helps in excluding polyarteritis nodosa.

While the etiology of TA remains obscure, some studies have suggested an underlying auto-immune process. Evidence in favor of such a process include: (1) Immunoglobulin deposition along the disrupted internal elastic membrane in involved vessels,[5] (2) an increased *in vitro* stimulation of lymphocytes by arterial wall antigen by patients with PMR when compared to controls[6] and (3) an increased incidence of HLA-8 in one small series (this has been associated with other auto-immune

syndromes such as auto-immune thyroiditis, myasthenia gravis, Sjogren's syndrome and chronic hepatitis).[7] Cell-mediated mechanisms have also received support in the literature.

Clinically the most frequently presenting complaints include headache, tender temporal arteries, jaw claudication, polymyalgia rheumatica, constitutional symptoms and visual symptoms. The headache is often nonspecific, but classically presents as an abrupt onset of pain localized to temporal area that may radiate to the scalp, face, jaw or occiput. It is of a continuous, boring quality with occasional episodic lancinating pain.[8] The pain may be worse at night and may be increased by exposure to cold weather. Paroxysms of pain may occur when trigger points are stimulated by hair brushing, lying on a pillow, or by the pressure of eyeglasses on the temples. Jaw claudication (secondary to facial artery occlusion) and the lingual Raynaud's phenomenon are considered the only pathognomonic features of TA.[9] Constitutional symptoms include fever (with patients occasionally presenting as an FUO),[10] anorexia, malaise, weight loss and depression. Loss of vision is the most frequent serious sequela of TA, with an overall incidence of 30-50%. However, less than 10% of patients present initially with blindness and most patients experience premonitory symptoms of several months' duration prior to their visual loss. The blindness is irreversible and contralateral eye involvement can occur in an interval as short as several weeks. Other ocular manifestations include diplopia, ptosis, amaurosis fugax, scintillating scotomata and photophobia. Unfortunately there are no symptoms, physical findings, or laboratory abnormalities that have predictive value in distinguishing those patients who develop ocular involvement. Polymyalgia rheumatica characterized by diffuse stiffness and aching in proximal muscle groups, most frequently involves the neck and shoulder girdle and less commonly involves the hip girdle, lumbar spine and the thighs. It is usually worse in the morning and may make the clinician think of rheumatoid arthritis; however, there is only a transient synovitis and very infrequent metacarpalphalangeal joint involvement. As opposed to polymyositis, there is no weakness and the EMG and muscle enzymes are normal as well as the absence of the distinctive muscle biopsy findings. Cumulative data among patients with PMR without signs or symptoms of TA, reveal that approximately one-third will have positive biopsy findings. Patients with positive biopsies were indistinguishable on the basis of age, sex or laboratory findings.[11] Uncommon symptoms of TA include limb claudication, aortic arch syndrome, abdominal pain, angina, carpal tunnel syndrome, acute hearing loss and CNS manifestations, including hemiplegia, cranial nerve palsies, vertigo, seizures, peripheral neuropathy and ischemic radiculopathy.[12]

The physical exam may be helpful in making the diagnosis of TA. Ocular exam may reveal ophthalmoplegias, evidence of visual loss or field cuts, and funduscopic abnormalities following blindness. The temporal arteries may reveal nodularity, decreased pulsation, tenderness, bruits and overlying erythema. The systemic nature of this illness manifests as pallor, cachexia, weakness and fever.

The sedimentation rate is almost always elevated with values frequently greater than 100mm./hr. There are, however, many documented cases of TA without elevated ESR's, and this should not prevent aggressive diagnostic and therapeutic intervention when the history or exam is suggestive. Other abnormal laboratory findings include anemia which is present in 30% of patients (usually normochromic or hypochromic), abnormal liver function tests (especially an elevated alkaline phosphatase which is felt to be due to a granulomatous hepatitis, and not a vasculitis), and elevated alpha and beta-2 globulins. Muscle enzymes, rheumatoid factor and ANA are usually negative or within normal limits.

Definitive diagnosis can only be made by biopsy. Arteriography may help in selecting biopsy sites, but cannot be used diagnostically since atherosclerosis may create changes indistinguishable from TA. Because of the presence of skip lesions, a 4-6 cm. biopsy of the temporal artery is recommended. When negative, and the clinical suspicion remains strong, other biopsy sites may include the contralateral temporal artery, facial artery or occipital artery. The question of routine temporal artery biopsy in all patients with PMR without evidence of TA is still debatable.

Corticosteroids are the mainstay of treatment. Symptoms quickly resolve, and ERS frequently falls, and further visual deterioration is abated once proper steroid treatment is instituted. Nonsteroidal anti-inflammatory agents are contraindicated because they are ineffective in preventing blindness while offering a false sense of security by reducing the ESR and constitutional symptoms. Initial therapy consists of 60-80 mg. per day of Prednisone or its equivalent for 4-6 weeks with a gradual reduction as the clinical picture and ESR allow. Recommended therapy is for a minimum of 1-2 years.[13] Alternate-day steroids have been shown to be significantly less effective than daily steroids.[14] An important point to keep in mind while managing these patients is the need for close and regular follow-up for a period of many years.

As I have attempted to illustrate, temporal arteritis is an important disease of the elderly that can present with a myriad of symptoms. Because the devastating catastrophe of blindness can be prevented with steroids, temporal arteritis should be considered whenever the clinical syndrome is even remotely suggested. Awareness of this common disease is important since physicians from such diverse fields as internal medicine, neurology, family practice, and surgery will frequently be confronted with this difficult diagnostic problem.

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Oncology

Stage II Carcinoma of the Endometrium

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Adenocarcinoma of the endometrium is now the most common gynecologic malignancy, with nearly 27,000 new cases being diagnosed each year. Approximately 10% to 15% of these cases will fall into the category of Stage II disease. On a world-wide basis, approximately one half of these patients will succumb to their disease.

Stage II carcinoma of the endometrium as defined by FIGO (International Federation of Gynecology and Obstetrics) denotes extension of the tumor to the cervix.[1] Since the management of Stage II disease is quite different from that of Stage I endometrial carcinoma, it is extremely important to do a careful differential curettage obtaining a separate endocervical specimen prior to any manipulation of the uterine fundus. The pathological interpretation of the endocervical material is critical, since carcinoma may be inadvertently brought down from the uterine fundus. At the very least, endocervical glands must be present in direct continuity with the carcinoma. In fact, recent data collected at the University of North Carolina and Duke University Medical Centers[2] showed a significantly

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worse survival when invasion of the cervical stroma was demonstrated in the pathological specimen. (See Table 1.) Due to this difference in survival, we currently do not consider patients to have Stage II disease unless stromal invasion is demonstrated either in the endocervical specimen or in the uterine specimen postoperatively. Homesley[3] in reviewing the Memorial experience in New York found no difference either in survival or incidence of lymph node metastases when the cervical involvement was detected preoperatively or found occultly in the uterine specimen. Therefore, when cervical involvement in the uterine specimen is noted, treatment should be altered to fall in line with that appropriate for Stage II disease, which will be defined later.

The managment of Stage II carcinoma of the endometrium still remains controversial, as can be seen from Table 2. Kottmeier[4] at the Radiumhemmet in Stockholm for many years advocated radiation therapy alone for these patients with approximately a 50% 5-year survival; however, Joelsson[5] at the Radiumhemmet utilizing a combined approach of surgery and radiation therapy achieved much improved survival approximating 80%, which is much in line with what most authors[6,7] in this country report for a combination of surgery and radiation. Some authors[8] still advocate radical hysterectomy and bilateral pelvic lymphadenectomy; however, this is rapidly becoming a less favored approach for this disease. This is primarily due to the patient population with this disease. (The patients are usually more elderly and frequently obese with hypertensive and/or diabetic disease.)

To define the optimal therapeutic approach to Stage II carcinoma of the endometrium we must look at the field at risk for this disease, which is illustrated in Figure 1. The uterus, of course, is at primary risk with both endometrial and cervical involvment and is usually removed surgically. Patients who are not surgical candidates may have the primary lesion treated with a combination of intracavitary and external radiation therapy. The ovaries are also removed surgically with carcinoma of the endometrium, as even with Stage I disease the incidence of metastasis to the adnexa approximates 10%, and one would expect it to be somewhat higher with Stage II disease. The parametria is at risk once the cervix is involved, and this is usually treated with a combination of intracavitary and external radiation therapy in a fashion similar to that for cervix cancer, only with somewhat less intracavitary radiation therapy.

The incidence of pelvic nodal metastases in Stage II disease approximates 30% and this increases to nearly 45% with poorly differentiated lesions of the endometrium involving both the fundus and cervix. This high incidence of lymph node involvement with Stage II disease requires that all patients receive external radiation therapy to treat this area. The para-aortic nodal involvement has not been assessed in the

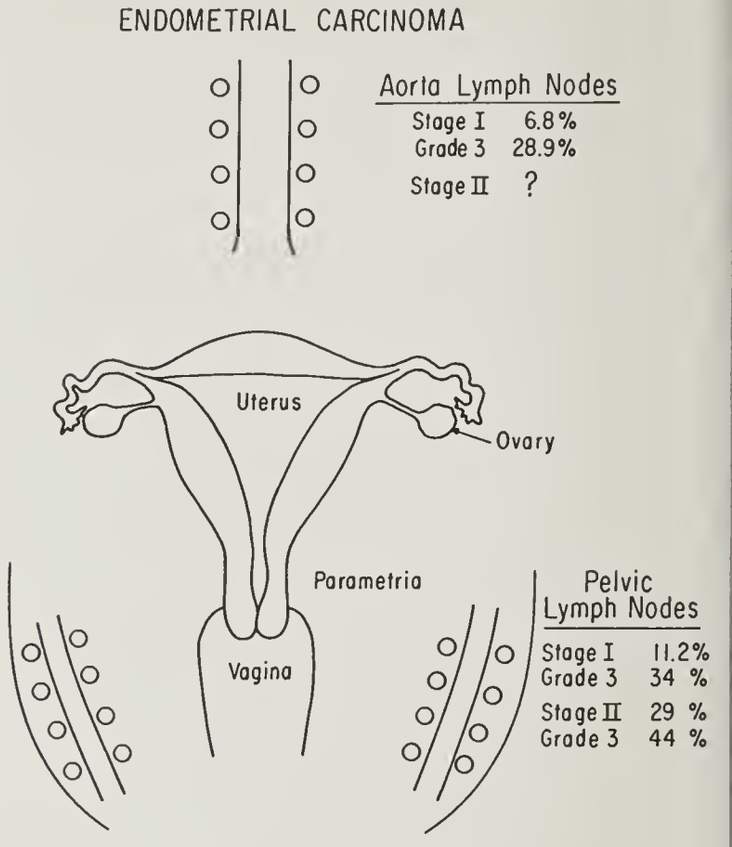


Figure 1
Morphologic pattern of endometrial carcinoma and incidence of nodal metastases.

Table 1
Endocervical Stromal Invasion
Survival - Three Years

Invasion of Cervical Stroma	37/78 (47%)
No Invasion of Cervical Stroma	29/39 (74%)

Table 2
Five Year Survival
Stage II Endometrium

	Treatment	Survival
Morrow, et. al.[12]	World Literature Summary	51%
Kottmeier[4]	Radiation Alone	53%
Lewis and Stallworthy[8]	Radical Hysterectomy and Bilateral Pelvic Lymphadenectomy	61%
Boronow[6]	Surgery + Radiation	71%
Thompson, et. al.[7]	Surgery + Radiation	80%
Joelsson, et. al[5]	Surgery + Radiation	80%

Table 3
Stage II Endometrium
Three Year Survival

RT + Surgery (73 pts.)	70%
or Surgery (10 pts.)	
RT Only (34 pts.)	29%
$p < 0.001$	

Table 4
Grade of Tumor and Three Year Survival

Grade	Radiation Therapy Alone	*Radiation Therapy and Surgery
I	7/13 (54%)	32/36 (89%)
II	1/9 (11%)	19/29 (66%)
III	2/12 (17%)	7/18 (39%)

**The ten patients who had surgery only are included in the radiation therapy and surgery group.*

literature with Stage II disease; however, adequate studies have been carried out with Stage I disease with an overall incidence of nearly 7%, increasing to nearly 30% involvement of the para-aortic nodes with poorly differentiated lesions. One would certainly expect this incidence to be greater with Stage II disease, especially in the anaplastic lesions. The treatment of patients with positive para-aortic lymph nodes is outlined later.

In carcinoma of the endometrium the upper vagina is at risk for recurrent disease. This approximates 14% with Stage I disease and would be somewhat higher for Stage II disease. This area is well encompassed by the combination of internal and external radiation which treats both the parametria and the pelvic lymph nodes.

A retrospective analysis of 117 patients with Stage II carcinoma of the endometrium was carried out at the University of North Carolina and Duke University Medical Centers to best delineate the optimal therapy for these patients.[2] As can be seen from Table 3 the majority of these patients were treated with a combination of surgery and radiation therapy. All patients received between 4,000 and 5,000 rads of whole pelvic radiation therapy and between 1,500 and 3,500 mg hours of intracavitary radiation (tandem and ovoids) followed at six weeks by a total abdominal hysterectomy and bilateral salpingo-oophorectomy. Optimal results were obtained with this combined approach with an overall survival of 70%.

The survival of only 29% for patients treated with radiation alone was attributed primarily to the inadequacy of the radiation in controlling pelvic disease. The increased incidence of poorly differentiated lesions in this group of patients contributed to the poor survival; although, as can be seen in Table 2, the results in the anaplastic lesions were improved when radiation and surgery were combined. All patients in this series had their intracavitary radiation therapy in the form of tandem and ovoids. If the uterus is not going to be removed, this is an inadequate means of intracavitary therapy for controlling pelvic disease. It has been well documented that Heyman packings are far superior to tandem and ovoids for achieving tumoricidal doses to the full uterine wall.[9] This would be particularly important in those patients with anaplastic lesions of the uterus where one would expect a high incidence of deep myometrial invasion which would mandate tumoricidal doses to the full thickness of the myometrium. In fact, Kottmeier's 5-year survival of 53% in patients treated with Heyman packings and external radiation therapy for Stage II disease certainly is far superior to the results obtained in our series of 29%.

The survival for patients treated with a combination of radiation therapy and surgery in our series of 70% compares quite favorably with series reported elsewhere.[5,6,7] It would thus appear that as long as the uterus is being removed the combination of external whole pelvis and intracavitary irradiation is adequate preoperative treatment for the primary tumor and also for the parametrial areas and pelvic lymph nodes. The role that Heyman packings might play in the preoperative treatment of patients with Stage II poorly differentiated carcinomas is yet to be defined.

The involvement of para-aortic lymph nodes in Stage I disease is well documented.[10] This area must be taken into consideration in planning therapy for patients with Stage II carcinoma of the endometrium, and all patients should have the para-aortic lymph nodes sampled at the time of definitive surgery.

We can now outline a treatment plan for patients with Stage II carcinoma of the endometrium. All patients should receive 4,500 rads of whole pelvic external irradiation and one intracavitary application with a tandem and ovoids for 50 hours or approximately 3,500-4,000 milligram hours. This should be followed at six weeks with a total abdominal hysterectomy, bilateral salpingo-oophorectomy, and para-aortic lymph node sampling. At the time of surgery, peritoneal washings should be obtained as data previously outlined[11] would indicate that this is a significant prognostic factor. If the peritoneal washings are positive, the patients postoperatively receive 15 millicuries of radioactive phosphorous intraperitoneally.

If the para-aortic lymph nodes are positive, 4,500 rads external irradiation is given to a field with an upper level of T12. All patients with positive para-aortic lymph nodes are also treated with adjuvant progestational therapy for one year. If the receptor site status has been evaluated and the tumor has been shown to be receptor site negative, then adjuvant chemotherapy with Adriamycin is given for six months.

Patients who do not have true endocervical stromal invasion documented at the time of differential

dilatation and curettage are managed as outlined previously for Stage I disease.[11] Patients who have occult cervical involvement found at the time of hysterectomy for Stage I disease receive postoperative whole pelvic radiation therapy, and if no preoperative intracavitary therapy was given, an intracavitary application with vaginal ovoids is added to the external irradiation. Patients who are not considered to be surgical candidates are treated with a combination of intracavitary Heyman packings (three times) and external irradiation to both paracervical areas and pelvic lymph nodes. This overall therapeutic approach should lead to 70%-80% 5-year survival for patients with Stage II carcinoma of the endometrium.

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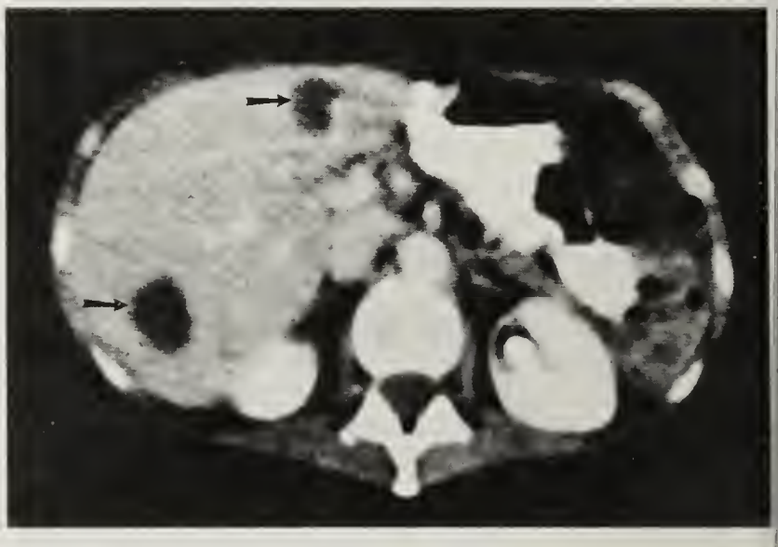
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Radiology

Case of the Month #46

Jay Post, M.D.
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Editors:
Jonathan Levy, M.D.
Gearld Pond, M.D.



A 52-year old female with known Hodgkin's disease had a two-week history of fever, chills, nausea, vomiting, and diffuse abdominal discomfort. During a prior hospitalization, recurrent episodes of *E. coli* speticemia were documented.

An abdominal CT scan was obtained. What are the abnormal findings?

What is the *most likely* diagnosis?

From: University of Arizona Health Sciences Center, Department of Radiology, Tucson, AZ 85724.

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Special Article

Attorneys' Requests for Patient Records

Roger W. Kaufman

1. Introduction:
Requests for Patient Records without the Patient's Authorization

Dr. Smith is a busy private practitioner. In the office mail one morning, he receives a notice of a deposition, which is accompanied by a subpoena duces tecum,[1] to bring medical records of his patient John Doe to a deposition. A cover letter from an attorney states that if the doctor will mail copies of the complete patient file, it will be unnecessary for him to appear at the deposition. In response to this common practice, Dr. Smith instructs his secretary to make copies and mail them to the attorney in order to avoid attending the deposition. Dr. Smith assumes that what he is doing is lawful because he is following the instructions of an attorney. The subpoena convinces Dr. Smith that release of his patient's records must be lawful.

What Dr. Smith does not realize is that in releasing confidential patient records without the knowledge or consent of the patient involved, he violates his patient's legal rights. And he may face serious penalties including having to pay his patient for injuries from wrongful disclosure of confidential information.[2] Nonetheless, many physicians each year improperly release patient's records.

The purposes of this article are to alert lawyers and physicians to problems with this common practice and to offer some suggestions.

From: Lewis & Roca Lawyers, One Hundred West Washington Street, Phoenix, AZ 85003. The author acknowledges the substantial help of Carole Clark in preparing this article which is general information and not legal advice. Search warrants are beyond the scope of this article and require separate consideration.

2. Privilege

Statutes creating the physician-patient privilege are designed to prevent the doctor from disclosing confidential information communicated by the patient or acquired in examining and treating the patient. They have been enacted in most states.[3] The rationale behind such laws is to encourage full disclosure to the doctor of everything which may have a bearing upon diagnosis and treatment and to preclude humiliation and embarrassment stemming from disclosure to others.[4]

The physician-patient privilege belongs to the patient, and must be claimed by the patient or someone authorized by law to do so on the patient's behalf.[5]

If the patient whose records are being sought is unaware of the request for confidential information, the doctor should obtain the patient's authorization to disclose privileged records or assert the privilege on behalf of the patient. The physician-patient privilege also extends to hospital records, and, in a recent Arizona case, the court held that when neither patient nor physician is a party to the legal proceedings, the hospital must assert the privilege.[6]

The privilege extends to communications between physician and patient obtained for purposes of treatment or possibility of treatment.[7] Knowledge gained by a physician through court-ordered examination has been held not privileged.[8] Dates of a patients' visits have also been held to be outside the privilege.[9] On the other hand, many hospital records and reports of medical staff committees are privileged.[10]

How will a physician know if the requested information is privileged? If the attorney's letter requests the complete patient file, it can safely be assumed that there is privileged information. If there is doubt, the privilege should be asserted.

It is a federal crime to release records of the identity, diagnosis, prognosis or treatment of any patient maintained in connection with the performance of any drug or alcohol abuse prevention function directed or assisted by the federal government.[11] These records can be obtained from a hospital or physician only if authorized by an appropriate court order granted upon a showing of good cause. This is different from a routine subpoena issued by a clerk. Federal law mandates that a *court* weigh the public interest and the need for disclosure against the injury to the physician-patient relationship, before allowing release of these records.[12] Penalties for violating this law include fines up to $5,000 for each offense.[13]

Only the patient has the right to waive the physician-patient privilege and the usual waiver is a written signed consent to the release of records. A physician may never waive for the patient. A physician should not even turn over records to the patient's own attorney without the patient's authorization.

3. The Scope of Discovery

Court rules of discovery are very broad. Unless otherwise limited by order of the court, parties may generally obtain discovery regarding any matter, not privileged, which is relevant to the subject matter involved in a pending lawsuit.[14] A physician may be required to attend a deposition, even though neither he nor his patient are parties to the litigation. If a subpoena duces tecum has been properly issued, the physician who feels that the requested records are confidential must protect himself from possible contempt charges. Although a subpoena duces tecum can be easily obtained from the clerk of the court without a judicial order, it carries with it the full force of the law.[15]

What protections does the law afford to a physician whose records have been requested for inspection? The courts have discretion in limiting the scope of discovery and can modify unreasonable subpoenas and issue protective orders against discovery.[16]

4. Reponses of Physicians

(a) If a consent form properly signed by the patient accompanies the attorney's letter, the physician may mail copies of the records without hesitation or further checking.

(b) If the patient is a party to the litigation, has a lawyer who has notice of the deposition, and does not object after reasonable time, he may in essence have waived the privilege and the physician may respond by mailing copies of that patient's records. The physician may rely on the patient's attorney to protect the patient's rights; however, *the physician should contact the patient in order to confirm that the patient's attorney had knowledge of the request and that no protective order is being sought.*

(c) On the other hand, if the patient is not a party litigant, he may be completely unaware that his records are sought. Short notice may also leave the patient and his attorney unaware. (Both circumstances often occur in criminal prosecutions where evidence concerning the victim's medical record is sought.) Physicians should not release records when the patient may not know of the request unless there has been proper authorization by the patient or there is a court order.

(d) The law requires that a subpoena be properly served.[17] *If the subpoena has been mailed and not served, it can be ignored.* Courtesy may dictate checking with the patient and the lawyer first. *If a subpoena has been properly served, the physician has at least two options.*

(1) He can appear at the deposition with the records but refuse to disclose them on the basis of privilege; or

(2) The physician may call the attorney who wants the records and ask for a signed patient release. If it is provided, the records can be mailed. If the lawyer refuses and threatens the doctor with legal sanctions for failing to comply with the subpoena, the doctor should then contact the patient's attorney. If necessary, the doctor can contact his own attorney who can, at the doctor's expense, seek a protective order or file a motion to quash the subpoena. If no one has obtained a court order by the time set for the deposition, the physician must appear. He may ask to adjourn the proceedings until an order is obtained.[18] A physician should neither ignore a properly served subpoena nor discuss or show anyone confidential patient records without proper authorization from the patient or a court.

Footnotes

1. A subpoena duces tecum is a document issued by the clerk of the court commanding the person to whom it is directed to produce the books, papers, documents, or tangible things designated therein. *16. A.R.S Rules of Civil Procedure 45(b).*
2. Courts have generally upheld the right of a patient to recover damages from a physician for unauthorized disclosure concerning the patient on the grounds that such disclosure constitutes an actionable invasion of the patient's privacy, breach of fiduciary duty or breach of contract. *See, e.g., Horne v. Patton,* 291 Ala. 701, 287 So. 2d 824 (1973); *Hammonds v. Aetna Casualty & Surety Co.,* 243 F. supp. 793 (N.D. Ohio 1965); *Clark v. Geraci,* 29 Misc. 2d 791, 208 N.Y.S.2d 564 (1960); *Hague v. Williams,* 37 N.J. 328, 181 A.2d 345 (1962). A Pennsylvania court has even permitted the husband of a patient to maintain suit against a doctor for threatened disclosure of medical information concerning his wife. *Clayton v. Bernstein,* 38 Pa. D & C 543 (1940) as cited in Horne v. Patton, supra. In order to avoid this type of exposure, physicians should use caution in releasing patient records in response to requests of attorneys.
3. 61 Am. Jur. 2d, Physicians, Surgeons, and other Healers, § 101. Arizona has enacted two statutes which deal with physician-patient privilege:

 A.R.S 12-2235 Doctor and patient

 In a civil action a physician or surgeon shall not, without the consent of his patient, or the conservator or guardian of the patient, be examined as to any communication made by his patient with reference to any physical or mental disease or disorder or supposed physical or mental disease or disorder or as to any such knowledge obtained by personal examination of the patient.

 A.R.S 13-4062(A) Anti-marital fact privilege; other privileged communications

 A person shall not be examined as a witness in the following cases:

 4. *A physician or surgeon, without consent of his patient, as to any information acquired in attending the patient which was necessary to enable him to prescribe or act for the patient.*
4. *Berry v Moench,* 8 Utah 2d 191, 331 P.2d 814 (1958).
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11. 42 C.F.R. § 2.1.
12. *Id.*
13. 42 C.F.R. § 2.14.
14. A.R.S. Rules of Civil Procedure § 26
15. A.R.S Rules of Civil Procedure §45(a) *State Farm Ins. Co. v. Roberts,* 97 Ariz. 169, 398 P.2d 671 (1965); *Ingalls v. Superior Ct.,* 117 Ariz. 448, 573 P.2d 522 (1977).
16. A.R.S. Rules of Civil Procedure § 26(c), § 45(b).
17. A.R.S. Rules of Civil Procedures § 45 (d).
18. It is necessary that court approval be obtained, rather than merely filing a motion. *Jolly v. Superior Ct.,* 112 Ariz. 186, 540 P.2d 658 (1975).

Radiology Continued

Diagnosis: Multiple Liver Abscesses

The CT scan revealed multiple discrete rounded areas of less density than liver, two of which are easily seen on the slice shown (arrows). The largest lesion measured approximatley 2.5 cm in diameter and was located in the right lobe.

In a patient with Hodgkin's disease, the clinical history and radiographic findings strongly suggest the diagnosis of multiple liver abscesses. Other diagnostic considerations include multiple cysts, lymphomatous involvement of the liver, and metastases. The CT density of the lesions was too high for cysts. The discrete nodular appearance mitigates against the diagnosis of lymphoma. The possibility of metastatic neoplasm was unlikely with the clinical history.

Most liver abscesses are seen by CT as sharply defined, low density areas. The density of these lesions is generally greater than that of water, but less than that of neoplasm or normal liver parenchyma. A large percentage of liver abscesses are found in the posterior aspect of the right lobe, for reasons thought to be related to the blood flow pattern within the portal venous system.[2]

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State of the Art

State of the Art Review: Adrenergic Bronchodilators

Brian Y. Shon, M.D.

Editor:
Eric H. Prosnitz, M.D.

Today there are an ever increasing number of bronchodilators for use in the treatment of asthma, bronchitis, and emphysema. In deciding which agents to use, the clinician must consider the properties of each and the optimum route of administration: oral, aerosol, subcutaneous, or intravenous. In this article the pharmacology of the adrenergic bronchodilators will be briefly reviewed and the use of these agents in the treatment of bronchospasm will be discussed.

Classification

The adrenergic bronchodilators include a large number of oral, parenteral, and aerosol preparations. Isuprel and epinephrine were the first drugs of this type to be used in the treatment of bronchospasm. Like these two agents, all adrenergic bronchodilators have a number of actions in addition to their bronchodilator effect. To account for the different actions of adrenergic agonists at different sites, the concept of alpha and beta adrenergic receptors was proposed over 25 years ago.[1] Stimulation of alpha receptors is associated with vasoconstriction, intestinal relaxation, uterine contraction, ureteral contraction, and pupillary dilation. Stimulation of beta receptors is associated with relaxation of blood vessels, intestine, uterus, ciliary muscle, and bronchi; an increase in cardiac rate and force of contraction; and excitation of skeletal muscle with tremor. More recently the beta receptors have been subdivided two types, into beta-1 and beta-2.[2] Beta-1 properties are primarily those of cardiac stimulation. Beta-2 properties are those of bronchial, uterine, and vascular smooth muscle relaxation, muscle glycogenolysis, and excitation of skeletal muscle.

Adrenergic agents can be classified according to their relative, alpha, beta-1 and beta-2 properties. For example, epinephrine stimulates alpha, beta-1, and beta-2 receptors. In contrast, isoproterenol stimulates only beta-1 and beta-2 receptors. The ideal adrenergic bronchodilator would be a pure beta-2 agonist. Recently, several compounds having strong beta-2 properties have been synthesized. These along with other frequently used adrenergic agents are listed in Table 1.

Epinephrine

Epinephrine, or adrenalin, was the first adrenergic agent to be used in the treatment of bronchospasm. It is available for subcutaneous administration and is also used in over-the-counter propellant inhalers (Primatene,® Bronkaid,® Asthmanefrin®). Epinephrine has approximately equal alpha and beta effects. Its use is limited by its cardiovascular side effects; its use is not recommended in patients with significant myocardial disease.

Epinephrine has an extremely short duration of action because it is rapidly metabolized and is taken up by sympathetic nerve endings. It is broken down by both

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Table 1
Adrenergic Bronchodilators

Drug	Actions	Usual Dose	Onset of Action	Peak Effect	Duration
Epinephrine Aerosol	α, β_1, β_2	0.2 mg/puff 2 puffs	1-5 min.	5 min. 90-120 min.	2-3 hours.
Subcutaneous		0.01 mg/kg	10-15 min.	1 hr.	< 4 hrs.
Ephedrine Capsules	α, (β_1, β_2)	15-50 mg. every 3 hrs.	1 hr.	2 hrs.	3-5 hrs.
Isoproterenol Aerosol	β_1, β_2	0.125 mg/puff 2 puffs	2-5 min.	5-30 min.	1-3 hrs.
Isoetharine Aerosol	(β_1), β_2	0.34 mg/puff	2-10 min.	15-60 min.	1.5-3 hrs.
Metaproterenol Aerosol	(β_1), β_2	0.65 mg/puff	2-10 min.	30-90 min.	1-5 hrs. (single dose) 1-2.5 hrs. (repeated doses)
Tablets	(β_1),β_2	10-20 mg. every 6 hrs.	30 min.	2-2.5 hrs.	4 hrs.
Terbutaline Tablets	β_2	2.5-5 mg. every 6-8 hrs.	30 min.	2-4 hrs.	4-6 hrs.
Aerosol		0.5-1.5 mg/puff	2 min.	1-2 hr.	4-6 hrs.
Subcutaneous	β_1, β_2	0.25 mg.	5-15 min.	30-60 min.	1.5-4 hrs.
Salbutamol	β_2	Not available in U.S.A.			
Rimiterol	β_2	Not available in U.S.A.			
Fenoterol	β_2	Not available in U.S.A.			

catechol-o-methyl transferase (COMT) and by monoamine oxidase (MAO). Epinephrine is inactive when given orally since MAO is present in the gastrointestinal tract.

Ephedrine

Ephedrine sulfate, like epinephrine has both alpha and beta properties. However, it is only a weak beta stimulant. It has a longer duration of action than epinephrine and is well absorbed orally since it is not metabolized by COMT or MAO. Its use as a bronchodilator is limited by its alpha effects. Urinary retention is common. It has a marked stimulatory effect upon the central nervous system. Because of this, it is often given in combination with a sedative drug. Usual dosage in adults ranges from 15-50 mg given three times a day. Tolerance to the drug has been reported to develop when doses in excess of 60 mg are given.[3] Ephedrine is commonly included in fixed combination with a sedative and a subtherapeutic dose of theophylline preparation.

Isoproterenol

Isoproterenol is an almost pure beta agent. It has equal beta-1 and beta-2 properties. It is used as an aerosol and as an intravenous solution. It is ineffective when administered orally because it is rapidly sulfated in the intestinal wall and broken down in the liver. Its main side effects stem from its beta-1 cardiac stimulatory properties. It is well known to produce arrhythmias and has been reported to cause myocardial necrosis when given in large doses.[4] "Paradoxical responses" or rebound bronchoconstriction have been reported to occur in small numbers of patients after intravenous and inhaled isoproterenol.[5,6] This may represent an idiosyncratic response. In persons demonstrating such a response other bronchodilator agents should be used.

Beta-2 Agents

A number of compounds demonstrating a high degree of beta-2 specificity have been synthesized. Beta-2 agents approved for use in this country include isoetharine, metoproterenol, and terbutaline. These

agents have virtually no alpha effects. Their beta-2 properties far outweigh their beta-1 actions. The ratio of their beta-l stimulating effect to beta-2 stimulating effect ranges from 1:3 to 1:6. This contrasts with the 1:1 ratio of isoproterenol. Thus, a greater bronchodilator effect can be achieved before significant cardiac stimulation occurs. Major side effects which appear are those of other beta-2 effects, most frequently skeletal muscle excitation manifest as tremor.

Isoetharine has been available for many years as an aerosol agent in combination with phenylephrine (Bronkosol®). It has some beta-2 specificity, but has significant beta-1 effects. Isoetharine is relatively short acting since, like isoproterenol, it is metabolized by COMT and MAO.

Metaproterenol is not metabolized by COMT. Thus, it is long acting. It has some selective beta-2 activity. It is available as tablets or as a metered dose aerosol. When used as an aerosol tachycardia may occur in 4-8% of patients. Its onset of action and peak effect occur later than that of isoproterenol. When taken orally side effects increase to as high as 17%.[7]

Terbutaline is even more beta-2 selective than metaproterenol, and is longer acting. It is available as a subcutaneous agent, an aerosol solution, and as a tablet. Its major side effect is beta-2 mediated muscle tremor. This is reported to occur in 20-30% of patients taking a 5 mg dose.[7] Fortunately, tolerance to this side effect occurs in the majority of cases.[8] Used as a subcutaneous agent, terbutaline appears to lose its beta-2 specificity, giving it little advantage over epinephrine.

Route of Administration

In choosing an adrenergic bronchodilator one must consider the route of administration. Many of these agents are available in preparations suitable for aerosol, oral, or parenteral use. Administration by inhalation has a number of theoretical advantages. A smaller dose is required, the onset of action is rapid, and there are fewer side effects when compared to intravenous or oral administration. When given by aerosol, about one-hundredth of the oral dose is required to produce equivalent amounts of bronchodilation. Only about ten percent of the inhaled dose reaches the lung with a pressurized aerosol. Approximately twenty percent reaches the lung with a nebulized aerosol.[9] The amount of drug required for nebulization is larger because about 90 percent of the drug remains in the nebulizer. An added advantage of aerosol administration is that it has been shown to prevent induced bronchospasm.[10] This has not been found when the same drug is given orally in a dose which produces the same amount of resting bronchodilation.

Intravenous administration also has a rapid onset of action. However, a higher concentration of drug has to be used and the incidence of side effects is higher. This is also true of subcutaneous administration. A potential advantage of systemic administration of bronchodilator is that it may affect the smaller airways more than when inhaled.

Oral administration has a slower onset of action. The incidence of side effects is between that seen with inhaled and intravenously administered medication. This mode of administration is applicable for maintenance therapy.

The duration of action of a bronchodilator medication is directly related to the amount of bronchodilator present at its site of action. Thus, a bronchodilator, when inhaled, has a shorter duration of action than when it is systemically administered. The duration of action of some of the more commonly used adrenergic bronchodilators is listed for both aerosol and systemically administered doses in Table 1.

Therapy

Adrenergic bronchodilators can be used in the primary treatment of bronchospasm or in combination with other bronchodilating agents such as the theophylline preparations. The key to any successful strategy is to fit the treatment to the situation.

When a patient has mild wheezing episodes he may be well served by symptomatic use of an over-the-counter pressurized aerosol inhaler containing racemic epinephrine. For more severe episodic wheezing an isoproterenol inhaler may be required. If the attacks are exercise induced or if an inciting agent is known, pretreatment with an inhaled agent one-half hour before exposure has been demonstrated to prevent attacks.[10] Inhaled metaproterenol is especially useful in this regard since it has a longer duration of action than other available aerosols.

For continuous treatment ephedrine, which is inexpensive, may suffice if the bronchospasm is mild. Otherwise one of the oral beta-2 agents should be used. Patient acceptance is enhanced if they are forewarned of side effects and if therapy is begun with a submaximal dose (e.g. 2.5 mg of terbutaline). After two weeks the dosage can be increased. In more severe cases an inhaled agent should be combined with the oral medication.

Treatment with both adrenergic agents and theophylline preparations is indicated if control of bronchospasm is not achieved with maximum doses of one or the other. There is evidence that adrenergic and theophylline bronchodilators have at least an additive effect.[11] Note that fixed combination drugs (e.g. Tedral®) are inappropriate in this situation, since they contain submaximal doses of theophylline and a weak adrenergic bronchodilator, ephedrine.

In emergency situations rapid onset of effect is of importance. Thus, parenteral agents or inhaled administration of adrenergic bronchodilators is indicated. Parenteral agents include subcutaneous epinephrine or terbutaline, and intravenous isoproterenol. Epinephrine should be used with extreme caution in patients with cardiovascular disease because of its alpha and beta-1 effects. Subcutaneous terbutaline is an alternative but has significant beta-1 activity. Intravenous isoproterenol has been advocated in

pediatric cases.[12] There is no reason that it cannot be used in adults as a carefully controlled drip (0.5-5 mg/min) as long as pulse rate can be maintained at less than 120. The use of inhaled agents in emergency situations is debated. However, recent data suggests that aerosols can be effective if they are preceded by a parenteral agent.[13] Nebulized aerosols should be used in preference over pressurized inhalers, since delivery of medication to the lung is twice as efficient. Nebulized isoproterenol can be used for a rapid effect. For a more prolonged effect terbutaline is most useful. Aerosolized terbutaline can be pushed as high as is necessary to achieve an effect (this can be as high as 2.5 ml of 1:200 solution) as long as it does not produce significant tachycardia. It is worthwhile to try an aerosol after the patient has received parenteral agents. If the aerosol is tolerated and produces clinical improvement, it can be continued.

As is true of any type of treatment, bronchodilator therapy should be assessed by following the patient's clinical status and objective measurements of pulmonary function. The latter can be obtained with an inexpensive spirometer or peak flow meter. It is not clear whether it is in a patient's best interest to aggressively treat asymptomatic bronchospasm. However, it is clear that treatment should at least be directed toward the goal of returning a patient to a comfortable level of function compatible with his lifestyle.

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Arizona Medicine

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Editorials

The Complete Physician

- On Becoming a Political Activist -

Talk about recurrent themes—listen to the following. At the September meeting of the Tucson Psychiatric Society, Henry Brosin, M.D., Past President of the American Psychiatric Association, got up and exhorted the psychiatrists to become involved in the political process. He talked about the ever encroaching regulations that impinge on our practice of medicine.

The next person to speak was Elliott Heiman, M.D., President of the Arizona Psychiatric Society. He told of the large volume of information he receives from the American Psychiatric Association concerning national legislation currently being considered. He asked for help from the membership in digesting this information, drawing up position papers, then writing legislators about the doctor's position.

The third speaker was Alan Levenson, M.D., Chairman, Department of Psychiatry, College of Medicine, at the University of Arizona. He told of the recent defeat of Senate Bill 1177 which would, had it passed, allowed psychiatric patients being treated in any federally-funded programs, (such as community mental health centers, medicaid, or medicare), to dictate their treatment and sue their psychiatrist if they did not follow their dictate.

I was the fourth physician to get up. I told my colleagues about the State of Arizona Board of Medical Examiners and its attempt to draw up guidelines to determine "an excessive fee."

These reports by two current office holders in organized medicine, a past office holder and a department chairman from the College of Medicine were not part of the planned program for that night. Our reports were spontaneous and reflected the concern we felt.

My point is physicians in leadership roles see involvement by the practicing physician in legislative matters as vital in today's practice of medicine.

The Arizona Medical Association provides several excellent ways for the physician to become actively involved in the political process.

One of these ways is the "Physician for a Day" program. This program enables you to spend a day at the state legislature serving as House Physician. In such a role all of the state legislators' offices are open to you. This is a wonderful opportunity to pay them a visit and chat informally. Maybe, with such contact, you might even find a few who will be a little more willing to listen to medicine's story.

Another way to participate is through the Arizona Medical Political Action Committee (ArMPAC). ArMPAC is financially independent from ArMA and anyone can join. The PAC solicits funds for the purpose of supporting political candidates who take responsible position on issues involving the medical profession and the patients we serve. All that ArMPAC asks from its members is money, time and effort.

A third way to become involved is through the Legislative Committee. The Legislative Committee is composed primarily of physicians. They come from all parts of the state, so there is good geographical representation. Specialty Societies, HMO's, the Osteopathic Association, medical students, and housestaff are represented as well. Members of ArMA and County Auxiliaries attend. Guests coming from the Department of Health Services, the Board of Medical Examiners, the Hospital Association, the Cancer Society, as well as similar organizations are also included.

The current chairman, Richard O. Flynn, M.D., is a past-president of ArMA, and served in our State House of Representatives for several terms. Therefore, he has seen it from "both sides."

The process of reviewing prospective legislation is a complex one. Annually the committee will review some 70 to 80 separate bills that will impact on the health and welfare of the citizens of Arizona.

This past year a new format was begun by Dr. Flynn. Committee members are given specific pieces of legislation to study and report on during the meetings.If special testimony is determined to be necessary on these pieces of legislation, the member is expected to attend the legislative hearings and testify on behalf of the Association. Generally, of course, the committee member assigned the task is from a geographical or specialty area most concerned with the legislation. Dr. Flynn and ArMA staff collaborate in making these assignments. This not only increases the interest of the Legislative Committee's members, but makes ArMA much more effective in its charge to "represent the Association in securing legislation in the interest of public health and scientific medicine, and in preventing the passage of, or securing the repeal of the legislation contrary to these interests."

When legislation is introduced that is of particular interest to a specialty society, or a group of physicians, the Legislative Committee involves the leadership of that society in the committee's activities. The following are two examples:

We have advanced legislation of special interest to the Pediatric Society by causing the Newborn Insurance Act to be adopted. The committee worked with the Psychiatric Society in developing and updating commitment procedures.

The standing committees of ArMA have been frequently asked to develop position papers. For example the Health Manpower Committee has developed position papers on nurse practitioners, Podiatry, and Chiropractic that have been used extensively in our legislative program. The Medical Education Committee has addressed the problem of mandatory continuing medical education. The Professional Committee has been instrumental in advancing children's auto safety restraint legislation on behalf of the Pediatric Society. Our Malpractice Insurance Crisis Committee has also worked actively with the Legislative Committee.

As always, the Board of Directors has the final authority over ArMA's committees. However, it has extended the power to the Legislative Committee to make quick decisions without the necessity of a Board meeting. When quick decisions are required, policy dictates that the Legislative Committee chairman communicate with the president or Executive Committee to discuss the issues and make a decision. These are the present practices and policies; they are under constant review by ArMA's officers and administrative staff.

Naturally, we always welcome suggestions on how we can be even more effective in speaking as the voice for all Arizona physicians.

How does one become a complete physician for the 1980's? By becoming politically active. The "Physician for a Day" program, involvement in ArMPAC, and/or ArMA's Legislative Committee are but three ways your Association provides you the opportunity to get involved.

John T. Clymer, M.D.
President, ArMA

Should we as Physicians Help Direct Hospital Growth?

It seems to me that non-profit hospitals give us a false sense of security in that they proclaim they are performing their function solely in "the public interest," but who determines what is in "the public interest"?

Hospitals have become very profitable businesses which are devoid of much regulation and as such find themselves in the enviable position of acquiring assets which may at times be at odds with what others would consider to be in "the public interest." Each hospital appears to desire to be the "mecca" for the best care. In order to give this level of care, each hospital wants to acquire the necessary assets to be so-classified.

Examples of such behavior are the proliferation of disease prevention seminars for the public, "dial-advice" medical service, separate buildings being built to offer preventive medicine seminars, "trauma center" designations being sought by many hospitals, and the proliferation of helicopter services with overlapping. Although these actions are basically good and do serve a useful function for the public, the key question is whether they are cost effective and whether they are truly needed. For the hospital, the answer is a resounding "yes!", but for the public, who ultimately pays for all of these services through health insurance premiums, the answer is probably not that clear-cut.

How many ambulance services, how many helicopters, how many trauma centers, how many dial-advice services, how many health preventive centers do we really need in our community? How many "meccas" do we really need? The hospitals are answering these questions for all of us in the guise of their non-profit status.

Shouldn't we as physicians have any input? I think the answer is "yes." Physicians in the past have brought change by exerting their influence as a collective group. I believe that the mushrooming costs in medical care are in part due to the expansion of these ancillary services which may or may not be in the public's best interest.

Each physician should question and then perhaps act in a concerted fashion to cull out the best and try to eliminate that which is redundant and nonessential. The public is looking to us for guidance, and we are at the present time awfully silent.

Marshall B. Block, M.D.
Editor

Hospice Care

Hospice is a relatively recent innovation in delivery of care to the dying and their families, in spite of its ancient origins. It is at once a concept of humane care as well as an integrated delivery system of medical, psychosocial and spiritual care. The modern hospice movement has been spearheaded by Cicely Saunders in England starting in the 1960's. The first hospice appeared in the United States in the mid 1970's initially following the British model. Since then, there are literally hundreds of hospices all over the country—many in planning stages and some fully operational. Hospice has become a movement which seems to have tapped deep felt needs around the care of the dying.

The hospice, as a system of care, has four coordinated components; an inpatient service, a home-care unit, a partial hospitalization unit combining inpatient and outpatient services, and a bereavement service.

The inpatient component is a special hospital for the care of the dying. Care is geared to providing the best quality of life for the patient and often requires sophisticated medical, diagnostic, and treatment skills. For instance, patients may require antibiotics for infections which cause weakness and withdrawal, or removal of pleural fluid, which compromises breathing, causing discomfort and perhaps confusion, or antidepressants to reduce depression which is often manifest in the elderly as confusion.

It seems best to have the hospice inpatient unit in a general hosptial setting because of the efficiency of integration of hospital services. In addition, hospice care requires an integrated team that specializes in care for the dying. This has become clearer as experience with hospice work has accumulated. The rigors of working with the dying require a staff that works together over time.

The home-care component attempts to help the family sustain the patient in the home. In fact, many patients are able to die at home relatively comfortably with the support of the home-care team. Usually, it is a nurse who makes the visits, but it may be any member of the hospice team, the general physician, the psychiatrist, the social worker, the chaplain, the dietician, or the physical therapist. Often, volunteers provide needed support and respite for the family by helping the family to care for the patient. Experience with a well-functioning home-care team has shown a decrease in inpatient length of stay. Patients are admitted now only when they are quite sick and the family is unable to care for them.

Bereavement services are an indispensable element in dealing with the dying and the family. Critical is the identification of those individuals at high risk for problems in the period after the death of the patient. Efforts are made to decrease the well-described morbidity noted in survivors in the bereavement period by follow-up, for up to a year in many hospices, after the death of the patient.

Usually, the population is an elderly one so that those on the team become experts in geriatric care. In addition, many of the patients in the hospice are usually those with end-stage cancer so that the team becomes expert in the management of cancer symptoms. Pain management using oral morphine or "hospice-mix," as well as antidepressants and psychological support, is an important element in the care of many dying cancer patients.

For a sense of success of one hospice, Hillhaven Hospice in Tucson, Arizona, the first freestanding hospice in the United States, took care of 51.3% of cancer deaths in Pima County in 1979.[1] Hillhaven Hospice has a close relationship with the University of Arizona and the College of Medicine. The hospice provides a brief clinical clerkship for medical students and a rotation for family practice residents. Members of the faculty of the University of Arizona and the College of Medicine have representation on the Hospice Advisory Board. One of the authors (SBS), who is a member of the College of Medicine Faculty, is also the Psychiatric Consultant to the hospice.

While cancer is a metaphor for death, and most of the literature on death and dying draws from clinical experience with cancer patients, cancer accounts for only

a quarter of all deaths. Perhaps as knowledge of treatment of the dying accumulates and the hospice concept gains more acceptance, more patients with other diseases will be referred to hospices.

In the meantime, the hospice is a teaching and research laboratory for the understanding of care of the dying and for the application of these principles to other settings. Third party payers are increasingly funding hospice care as they learn about its economic feasibility. This system of care will then become available to more patients and their families.

There is much that is fresh and new in this system of care for the dying patient and the family. There are, however, many unanswered questions such as,who is best suited for this type of care. The answers will emerge with more experience and research.

Stephen B. Shanfield, M.D.
Assistant Professor
Department of Psychiatry

Louis J. Kettel, M.D.
Dean, College of Medicine

1. Corrected to exclude deaths of patients under 15 and deaths from hemopoietic disease.

Auxiliary Highlights

Maricopa County

Innovative and effective are the best words to describe Maricopa County Medical Society Auxiliary.

It is nationally-known for creative health education projects such as the GEMS baby-sitting course, the nutrition puppets, and the Catastrophe Cottage home safety program which have been copied by medical auxiliaries across the country.

It raises huge sums of money for its philanthropies. In a report to Maricopa's 1,219 members, 1979-80 President Jeanne Weiland listed $4,432 for the Valley Center for the Deaf, a research grant of $10,849 to the Juvenile Diabetes Foundation; more than $9,000 to AMA-ERF; almost $2,200 in scholarships; $3,000 to the American Cancer Society to buy "Early Start to Good Health" kits for grade schools.

Maricopa's members, usually wearing the distinctive yellow work aprons that have become their trademark, also presented programs on breast self-examination, worked as volunteers at the Health Fair, conducted a Medical Market Junque and Rummage Sale, distributed immunization record booklets to doctors' offices, and sponsored the second annual "Emergency Run" which attracted more than 700 runners.

No wonder then, that Maricopa's outstanding community service throughout the years was recognized by the Hon Kachina Award of Luke's Men.

What's ahead for 1980-81? President Jan Lisboa says the "Early Start" program is off to a great beginning in Maricopa County. More than 85% of the 292 schools contacted have said they want the health education program kits for their kindergarten through third grade classes.

This year's GEMS course was held early in June, the first week of summer vacation. The revised GEMS Handbook was used for the 1,298 registrants. Mary Ellen Malone and Donna Asendorf head the GEMS committee.

The Auxiliary's October general meeting will be held at Paradise Valley Country Club and the program will feature holiday ideas for food and decorating. Tommy Moraca is making plans with her committee for a holiday coffee, small holiday boutique and shopping spree December 9 at the home of Mary Zahn.

The Maricopa Auxiliary will sponsor and underwrite a day-long program on "Rape, Assault, Incest and Family Violence." It will be geared to reach the average person in the community and will stress how to avoid being caught in these situations, methods of self-protection, and where victims can turn for assistance. The program will provide information about post-trauma counseling, police routine in these cases, and the county attorney's victims' assistance program. There'll be a frank discussion of forensic examination procedures and the hospitals equipped to handle these cases will be listed. Two of Phoenix's leading television personalities will moderate the panels—Mary Jo West of Channel 10 and Linda Alvarez of Channel 12.

The third annual Emergency Run is set for the first Saturday in February. Ann Smith heads the run committee and Helen Hanss will serve as vice-chairman.

Catastrophe Cottage and the home safety program are being upgraded by Fran Montasir and her committee. They're aiming for a more balanced program, with films or slides on poison control and fire prevention and safety.

The nutrition puppets will stress the importance of exercise and fitness as well as a proper diet when they go out to the schoolrooms of Maricopa County.

This Auxiliary is not content to rest on its laurels.

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ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, August 8, 1980, a quorum being present, convened at 6:50 p.m., John T. Clymer, M.D., President and Chairman, presiding.

Resolution 1-80
Appointment of Committe

The committee reviewed and discussed Resolution 1-80 adopted by the House of Delegates on April 25, 1980, regarding the appointment of a committee to determine the desirability of eliminating County-State unified membership and it was determined to take no action at this time, pending the Board's action on 8/9/80 relative to the calling of a special meeting of the House of Delegates for voting on the question of unified membership between ArMA and the AMA.

Review of Board of Directors' Agenda for 8/9/80

The agenda for the Board of Directors' meeting to be held August 9, 1980, was discussed, item by item, with additions and changes being made.

October Meeting

It was determined by the committee that, for convenience of the members, the October Executive Committee meeting would be held in Tucson on Saturday, October 4, 1980, at 8:00 a.m., immediately preceding the meeting scheduled for the Board of Directors.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, August 9, 1980, a quorum being present, convened at 10:10 a.m., John T. Clymer, M.D., President and Chairman, presiding.

Cloud Mailgram

Mailgram from Daniel T. Cloud, M.D., dated August 6, 1980, conveying his thanks for the Board's help and support in the recent AMA election. *Received.*

The Unified Membership Crisis

The Board considered the recommendation of the Executive Committee made during meeting on June 27, 1980, that a special meeting of the House of Delegates be called for the purpose of again considering the question of unified membership between AMA and ArMA. A lengthy discussion ensued, during which the Board received legal counsel's comments on the unified membership issue, together with like comments and opinions from several of the county societies, correspondence relating to the effect of unified membership on group employed physicians, and various pertinent statistics and fact sheets. Following a thorough review and discussion of all the supporting data,

It was moved and carried that a special meeting of the House of Delegates be called for the purpose of voting on the question of unified membership between the American Medical Association and the Arizona Medical Association, and that a special committee be immediately established to educate the Delegates on all aspects of this matter.

Auxiliary Activities Report

Joyce Newman, President of the ArMA Auxiliary, reported to the Board regarding some of the goals for the Auxiliary's 51st year, which included performance of good communication, assistance in school health education and programs on cost containment. Mrs. Newman also advised that seven delegates had attended the recent AMA convention, where they assisted as hostesses for Dr. Cloud's receptions, and during which convention Mrs. Ella Kimball was elected Western Regional Vice President.

ArMA Auxiliary letter of thanks dated 5/1/80. *Received.*

Board of Directors

AMA Liaison Committee on Medical Education

The Board reviewed a request for recommendation for nomination of a public member for the AMA Liaison Committee on Medical Education and

It was moved and carried that, subject to their consent to act, the Arizona Medical Association submit the names of Sandra O'Connor and Dr. Taylor Hicks as its nominations for membership on the AMA Liaison Committee on Medical Education. If neither is willing to serve the president shall have discretionary power to nominate any other individual he feels possesses the requisite qualifications in order to meet the August 15, 1980, deadline.

October Board Meeting

The Board was advised that the October 4, 1980, meeting of the Board would be held at the College of Medicine in Tucson, beginning at 10:00 a.m., followed by luncheon with the department heads of the College of Medicine, followed at 2:30 p.m. by an Official Family Briefing featuring Lowell H. Steen, M.D., Chairman, Board of Trustees, and Daniel T. Cloud, M.D., the newly-elected President-Elect of the AMA, including ArMA committee chairman and specialty society representatives.

Board of Medical Examiners

The current roster of the Board of Medical Examiners, together with notification of the appointment of Mario P. Valdez, M.D., to that Board. *Received*

Board of Optometry

Notification of appointment of Louis J. Rosenbaum, M.D., to the Board of Optometry. *Received.*

Executive Committee

Ratification of Minutes

It was moved and carried to approve and ratify the minutes of the meetings of the Executive Committee held on April 23, 1980, May 30, 1980, and June 27, 1980.

Membership Classification Changes Approved

Maricopa County Medical Society

1. Meyer Markovitz, M.D. - Active to Associate -Account Retirement - Effective 7/1/80 - Dues Exempt.
2. David C. James, M.D. - Active to Associate - Account Retirement - Effective 8/9/80 - Dues Exempt.

Pima County Medical Society

1. Clarence L. Clark, M.D. - Active to Associate -Account Retirement - Effective 8/9/80 - Dues Exempt.
2. DeArmond Lindes, M.D. - Service to Active - Account Out-of-Service - Effective 8/9/80.
3. Edward J. Nagoda, M.D. - Active to Associate -Account Retirement - Effective 8/9/80 - Dues Exempt.
4. Andrew S. Tedesco, M.D. - Resident to Active -Account Completed Residency - Effective 8/9/80.

Finance Committee

Arizona Perinatal Trust

The Board considered the action taken by the Finance Committee, in meeting on July 9, 1980, and of the Maternal & Child Health Care Committee, in meeting on May 24, 1980, regarding a request for funding by in-kind support to the Arizona Perinatal Trust. During a discussion of the matter, the Board was presented with updated information regarding the formalization of the Trust by Harold D. Gabel, M.D., and Ted F. Warner.

It was moved and carried that, when the Arizona Perinatal Trust is legally established, the Arizona Medical Association Foundation convey title to the furniture and equipment and, in addition thereto, convey title to any and all computer equipment, including radios and terminals, procured for and

being used by the Arizona Perinatal Program.

It was moved and carried to refer back to the Finance Committee the matter of providing accounting services to the Arizona Perinatal Trust, said matter to be reconsidered in light of the new structure intended for management of the trust when established and that a final recommendation of the finance committee be made prior to the October 4th meeting of the Board of Directors.

It was moved and carried that, in the interim, from August 31, 1980, until October 4, 1980, the Association would continue to provide accounting services up to the sum of $1,000.00 per month at no charge to the Arizona Perinatal Trust.

Financial Statement

Financial Statement for period ending 6/30/80. *Received.*

Physician Rehabilitation Committee

It was moved and carried to confirm the appointments of Thomas W. Edwards, M.D., and Judith Reid, M.D., to the Physician Rehabilitation Committee for the terms 1980-83.

Scientific Assembly Committee

1981 and 1982 Annual Meetings

The Board considered the advantages of changing the annual meeting dates for 1981 and 1982 to a later time in order to take advantage of "off-season" rates at the hotel and

It was moved and carried approving the dates of May 20-24, 1981, for the holding of the 1981 Annual Convention at the Pointe Resort in Phoenix.

It was moved and carried approving the dates of May 26-30, 1982, for the holding of the 1982 Annual Convention, subject to approval of the Scientific Assembly Committee during its meeting scheduled for August 16, 1980.

Change in Meeting Format

It was moved and carried to change the format of the business portion of the 1981 Annual Meeting to the following:

Wednesday, 5/20 - 10 A.M. - First House of Delegates Meeting

Wednesday, 5/20 - 2:00 P.M. - Reference Committees

Wednesday, 5/20 - 7:00 P.M. - Caucus Meetings

Thursday, 5/21 - 8:00 A.M. - Second House of Delegates Meeting

Articles of Incorporation and Bylaws Committee

Establishment of Standing Committee

The Board considered the recommendation of the Articles of Incorporation and Bylaws Committee made during meeting on July 12, 1980, regarding the establishment of that committee as a standing committee of the Association.

It was moved and carried to introduce to the House of Delegates during its 1981 Annual Meeting a resolution to establish the Articles of Incorporation and Bylaws Committee of this Association as a standing committee, said Resolution to read as follows:

Resolved, That Chapter VII, Section 4, of the Bylaws of the Arizona Medical Association be amended by the inclusion of the following:

(a) Articles of Incorporation and Bylaws:—This committee shall consist of a chairman and at least nine members. All members of this committee shall have been members of the Association for at least five years prior to their appointment.

This committee shall respond to resolutions of the House of Delegates by reporting it deliberative opinion and/or preparing proposed amendments to the Articles of Incorporation and Bylaws. The committee shall respond to directives by the board by reporting its deliberative opinion and/or preparing proposed amendments. All proposed amendments prepared in response to House or Board directives shall be presented to the Board of its consideration.

This committee may (Chapter X, Section 1) propose amendments for introduction to the House of Delegates.

Reorganiation of the House of Delegates

The Board discussed at great length a reorganization of the House of Delegates to include specialty society participation and considered the recommendations of the Articles of Incorporation and Bylaws committee as to the bylaws changes which would be necessary to bring this about. Following such discussion and review,

It was moved and carried to introduce to the House of Delegates during its 1981 Annual Meeting the following resolution which would provide for specialty society representation in the House of Delegates:

Resolved, That the Bylaws of the Arizona Medical Association, Inc.,be amended as follows:

"Chapter VIII
House of Delegates

"Section 1. Composition of House; Meetings:—The House shall constitute the voting body of the Association and shall be composed of the elected delegates of the county societies, **Elected delegates of the specialty societies,** elected Delegates of each duly constituted chapter of the American Medical Student Association in Arizona, elected Delegates of the duly constituted chapter of the Arizona Medical Assocation Housestaff Section, and members of the Board. Delegates who are thereafter elected as officers do not by such election lose their status as voting Delegates in the House. The past presidents of the Association shall be ex-officio members of the House of Delegates without the right to vote.

"The House shall meet at least once a year at the time of the Annual Meeting. In addition, special meetings of the House may be held at any time, upon at least six weeks' notice thereof to the Delegates, at the call of the Board, or upon the call of twenty Delegates *(representing at least eight)* **with representation from at least seven** county societies.

"Section 2. Number of Delegates:—

"(A) County Societies. Each county society shall be entitled to representation in the House by two Delegates, or their alternates, for the first twenty active members or fraction thereof, and one additional Delegate for each additional twenty active members or major fraction thereof, as determined on October first preceding the Annual Meeting.

"(B) Specialty Societies: A state specialty society shall be entitled to representation in the House of Delegates by one Delegate and alternate who shall be members of the Association if (a) The society has a national board recognized by the American Board of Medical Specialties, (b) A minimum of twenty members practicing in Arizona, the majority of whom must be members of the Association, (c) The activity of the specialty society is manifested by an existing organization or structure exhibiting a slate of periodically elected officers, an established constitution and bylaws, and a frequency of meetings at least once a year. If there is more than one qualifying society for a specialty, the Board shall determine the representative society. A specialty society shall have an additional Delegate and alternate for each additional 150 members of the society who are members of the Association, as determined October first preceding the Annual Meeting.

"(C) Housestaff Section; Student Association. Each duly constituted chapter of the Arizona Medical Association Housestaff Section and each duly constituted chapter of the Arizona Medical Association Housestaff Section and each duly constituted chapter of the American Medical Student Association in Arizona shall each be entitled to representation in the House by two Delegates or their alternates.

"Section 3. Election of Delegates; List thereof:—Sufficiently in advance of the Annual Meeting, each county society, **each specialty society,** The Arizona Medical Association Housestaff Section, and each duly constituted chapter of the American Medical

Student Association in Arizona shall elect Delegates and an equal number of alternates to represent it in the House. In the absence of any Delegate, *(his)* **the** alternate may vote in *(his)* **the Delegate's** name on any question before the House. No alternate shall be eligibe for election of office, but *(he)* may be appointed to membership on House committees in the absence of the Delegate. The secretary of each county society, **each specialty society**, *(and of)* the Arizona Medical Association Housestaff Section and each chapter of the American Medical Student Association shall send the list of such elected Delegates and alternates to the Secretary of the Association not later than two months before the Annual Meeting. Representation in the House shall be contingent upon compliance with this provision. A member to be seated must present evidence at the time of *(his)* appearance at the House *(,)* of *(his)* official election by *(his)* **the** county society, **specialty society**, *(or)* the Arizona Medical Association Housestaff Section, or chapter of the American Medical Student Association.

"Section 7. Quorum; Minimum Requirements for Vote: *(Fifteen)* **Twenty** Delegates, or their alternates, *(representing)* **with representation from** seven county societies shall constitute a quorum, and unless otherwise specified herein, a majority vote of the quorum shall be required for action."

(All italicized words represent deletions; all bold faced words represent new material)

Reports of ArMA Delegates

The Board received the reports of Drs. Chisholm, Dudley and Sattenspiel regarding the Annual Meeting of the AMA held July 20-24, 1980, in Chicago.

Correspondence

Letters of Appreciation

Letter from AMSA dated 4/24/80 and letter from American Board of Internal Medicine dated 6/25/80. *Received.*

Letter of Resignation

The Board reviewed the letter of resignation dated 7/21/80 from Roy S. Weinrach, M.D., as Arizona delegate to the U.S. Pharmacopeial Convention and it was determined to accept the resignation with a note of thanks and appreciation to Dr. Weinrach and that Dr. Clymer, as President, would make a selection of a physician whom he felt would best qualify as a representative of ArMA at future conventions.

Other Business

Council of Professions

The Board was informed that Joseph W. Hanss, M.D., has agreed to represent ArMA on the Council of Professions, replacing Stephen R. Stein, M.D.

Future Directions for Medical Education

Letter from James Sammons, M.D., Executive Vice President of AMA, requesting nominations to task forces on "Future Directions for Medical Education," dated 7/9/80. *Received.*

Western Physicians Purchasing Association

Letter of 7/14/80 from John F. Kahle, President of the WPPA. *Received.*

Press Release

Press Release dated 7/31/80 regarding recent scholarships granted by the Association. *Received.*

Maternal and Child Health Care Committee

It was moved and carried to confirm the appointment of Herbert L. Winograd, M.D., to the Maternal and Child Health Care Committee for the term of 1980-83.

Comings & Goings

College to Expand Membership Development

Fellows of the American College of International Physicians, Inc. have recommended to the College's Board of Trustees an expanded membership development program to recruit more interested physicians into the College.

The ACIP was organized in 1975 as a non-profit participatory Fellowship for physicians educated in medical schools abroad and in the United States, who are licensed and practicing in the United States. The Fellowship is open to all physiciasn who wish to join their colleagues from many nations around the world in a cooperative endeavor to shape policies for the future of medicine in the United States and around the world in the fields of medical education, research, ethics, and international health activities.

The College provides a forum for the discussion of issues of vital interest to international physicians and international medicine. It is actively working to help create a climate conducive to the development of the full potential of all international physicians now practicing in the American continent and is helping formulate the national interest in international health activities because of the unique internationality of its Fellowship and its expertise in the health needs of countries abroad.

Fellowship information and application forms can be obtained by writing the College's office at 3030 Lake Avenue, Fort Wayne, Indiana, or by calling (219) 424-7414.

Antonio B. Donesa, M.D., Executive Director
American College of International Physicians

It was recently announced that MacDonald Wood, M.D., Phoenix, Arizona, has been selected to be the state chairman for "Physicians for Regan/Bush."

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

OCTOBER

Pacific Coast Fertility Society Symposiums
October 16-19, 1980. Mountain Shadows Resort, Phoenix, Arizona. Sponsor: Pacific Coast Fertility Society. Contact: Dee Davis (213) 937-5514. Approved for 17 hours of category 1 credit.

Management of Common Neurologic & Neurosurgical Problems
October 17-18. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucosn, AZ 85724. Approved for 12 hours category 1 credit.

Practical Aspects of Infectious Disease in Obstetrics and Gynecology
October 18, 1980. U. of A. Health Sciences Center, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Department of Obstetrics and Gynecology U of A Health Sciences Center, Tucson, AZ 85724.

Family Dynamics in Medicine (Annual Doctor/Spouse Lecture)
October 21, 1980. Desert Samaritan Hospital, Cafeteria West End, Sponsor: Desert Samaritan Hospital. Contact: Office of CME, Desert Samaritan Hospital, 1400 S. Dobson Rd, Mesa, AZ.

Advanced Cardiac Life Support Program (Provider)
October 24-25, 1980. Arizona Medical Assn. offices. Sponsor: A.C.E.P., Arizona Affiliate - American Heart Assn., & ArMA. Contact: Carol Turner, Arizona Medical Asssn., 810 West Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Perinatal Update
Arizona Perinatal Trust
October 24-25, 1980. SunBurst Hotel, Scottsdale, AZ. Sponsor: Arizona Medical Association, Inc. Contact: Cathy Gunn, Arizona Perinatal Trust, 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 15 hours of category 1 credit.

Learning Disabilities: New Direction for the Physician
October 25, 1980. Sheraton-Scottsdale Inn, Scottsdale, Arizona. Sponsor: Good Samaritan Hospital. Contact: Mrs. Karen Iversen, Arizona ACLD Chairperson, 5211 E. Orchid Lane, Paradise Valley, AZ 85253. (602) 959-3192. Approved for 6 hours of category 1 credit.

Practical Management of Multiple Sclerosis
October 25, 1980. Phoenix, Baptist Hospital, Phoenix, AZ. Sponsor: Phoenix Baptist Hospital. Contact: Ruth Lee (602) 277-7441.

Poisoning and Drug Overdose Workshop
October 25, 1980. Arizona Health Sciences Center. Sponsor: University of Arizona College of Medicine & Arizona Poison Control System. Contact: Office of Continuing Medical Education, Attn: Faith Carlisi-Smith, U of A Health Sciences Center, Tucosn, AZ 85724. Approved for 8 1/4 hours of category 1 credit.

Neuro-Linguistic Programming
October 25 & 26, 1980. Aloha Hotel, Phoenix, Arizona Sponsor: Communication Dimensions, 1615 West Lewis, Phoenix, Arizona. Contact: Jeff Hatch, (602) 258-1702 or Judy Brieger (602) 993-2575.

Prospective Medicine & Health Hazard Appraisal: State of the Art
October 29 - November 1, 1980. Arizona Inn, Tucson,Arizona. Sponsor: University of Arizona College of Medicine and The American College of Preventive Medicine. Contact: Society of Prospective Medicine, c/o Sabina Dunton, P.O. Box 43338, Tucson, AZ 85733. Approved for 20 hours of category 1 credit.

7th Annual Fall Orthopedic Lectureship
October 30-November 1. Maricopa County General Hospital, Phoenix, Arizona. Sponsor: Maricopa County General Hospital. Contact: Department of CME, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 6 hours of category 1 credit.

Microvascular Surgery
October 31, 1980. Walter O. Boswell Memorial Hospital, Sun City. Sponsor: Walter O. Boswell Memorial Hospital. Contact: Office of CME, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ. Approved for 1 hour of category 1 credit.

NOVEMBER

Cardiology Symposium: Recognition & Management of Ventricular Arrythmias in Practice
November 1, 1980. Adams Hotel, Phoenix, Arizona. Sponsor: Maricopa County General Hospital, Dept. of Internal Medicine. Contact: Chairman, Dept. of Medicine, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008 (602) 267-5351. Approved for 6 hours category 1 credit.

Poisoning and Drug Overdose Workshop
November 1, 1980. Safari Resort Hotel, Scottsdale. Sponsor: University of Arizona College of Medicine & Arizona Poison Control System. Contact: Office of Continuing Medical Education, Attn: Faith Carlisi-Smith, University of Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 8 1/4 hours of category 1 credit.

A Course in Gynecological Laparoscopy
November 3-5, 1980. Good Samaritan Hospital, Auditorium, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: Office of CME, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ. Approved for 18 hours of category 1 credit.

Personal Instruction Course
In Intra & Extra Capsular Cataract Extraction with IOL Implantation
November 5-8 and 19-22. Sponsor and Contact: The Eye Foundation, Ronald W. Barnet, M.D., Medical Director, 421 N. 19th Street, Ste 105, Phoenix, AZ 85006.

Clinical Seminar Series "Endocrinology"
November 8. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours of category 1 credit.

Fact Sheet on Alcohol and Medicalton Abuse Workshop
November 8, 1980. Church of the Beatitudes, Phoenix, AZ. Sponsor: Alcoholism Committe, Junior League of Phoenix. Contact: Cindy Heckel, Vice Chairman, Alcoholism Committee, Junior League of Phoenix, Inc., c/o 4113 E. Dahlia, Phoenix, AZ 85032.

Diabetes Mellitus (Current Concepts)
November 12, 1980. Boswell Hospital, Room C-119, Sun City. Sponsor and Contact: Martha R. Newby, Dir. of Education, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for 1 hour of category 1 credit.

Medical Society of the United States and Mexico
November 12-15, Guadalajara, Jalisco, Mexico. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Exec. Secy., 3161 N. Pantano Road, Tucson, AZ 85715.

Dean's Clinical Rounds
November 13. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Lolly Dwan, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Rocky Mountain Clinical Geneticists
November 14, 1980. Tempe, Arizona. Sponsor: Genetics Center of Southwest Biomedical Research Institute. Contct: Joan Holman, The Genetics Center, 123 East University Drive, Tempe, AZ 85281.

American College of Physicians Regional Meeting
November 14-15, 1980. Arizona Biltmore Hotel, Phoenix, AZ. Sponsor: U of A College of Medicine. Contact: Office of Continuing Medical Education and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 9 hours of category 1 credit.

Arizona Pediatric Society
November 14-16. Westward Look Resort, Tucson. Sponsor: U of A College of Medicine & Arizona Pediatric Society. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 11 hours of category 1 credit.

"Risk Managment For Safety Professionals"
November 17. Student Union-U of A, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center for Occupational Safety and Health, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

Geriatrics Conference
November 20-21. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours category 1 credit.

The Legal Hurt of Chronic Pain
November 20, 21, 1980. The Mountain Shadows Resort, Phoenix, Arizona. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator. St. Luke's Hosptial, 525 N. 18th Street, Phoenix, AZ (602) 258-7373, Ext. 311. Approved for 14 hours of category 1 credit.

Comprehensive Review in Toxicology
November 20-22. Sheraton-Pueblo Inn, Tucson. Sponsor: Denver Institute of Clinical Toxicology. Contact: CRIT, c/o Peter Bryson, M.D, Emergency Department, Denver General Hospital, 8th Ave. & Cherokee St. Denver, CO 80204. Approved for 15 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
November 21-22, 1980. Arizona Medical Assn., Phoenix, AZ. Sponsor: ACEP, Arizona Affiliate-American Heart Assn., & ArMA. Contact: Carol Turner, ArMA, 810 West Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Thrombolytic Therapy
November 25, 1980. Desert Samaritan Hosp., Meas, AZ. Sponsor and Contact: Office of CME, Desert Samaritan Hospital, 1400 S. Dobson, Raod, Mesa.

Collective Review - Abdominal Aneurysm Cases
November 25, 1980. Walter O. Boswell Memorial Hospital, Sun City. Sponsor and Contact: Office of CME, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd.Sun City, AZ.

DECEMBER

State of the Art in Critical Care Medicine
December 2, 1980. Desert Samaritan Hospital. Sponsor and Contact: Desert Samaritan Hospital, Office of CME, 1400 S. Dobson Road, Mesa, AZ.

Controversies in the Management of Coronary Heart Disease
December 3 & 4, 1980. Los Angeles Hilton Hotel, Los Angeles, California. Sponsor: Lyman A. Brewer III International Surgical Society. Contact: The Secretary-Treasurer, Lyman A. Brewer, III International Surgical Society, 1720 Brooklyn Ave., Box 137, Los Angeles, CA 90033. Approved for 14 hours of Category 1 credit.

2nd Annual Sports Medicine Symposium
December 3-6. Marriott Hotel, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Faith Carlisi-Smith, Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

International Congress on Ericksonia Approaches to Hypnosis and Psychotherapy.
December 4-8, Phoenix, Arizona. Sponsor: Maricopa County General Hospital and The Milton H. Erickson Foundation, Inc. Contact: Jeffrey K. Zeig, Ph.D. The Milton H. Erickson Foundation, Inc. 1935 East Aurelius Avenue, Phoenix, Arizona 85020. Approved for hour per hour category 1 credit.

Occupational Medicine for the Primary Care Physician
December 6, 1980. Arizona Health Sciences Center, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724

Welding Safety and Health for the Mineral and Construction Industry
December 12, 1980. Marriott Hotel, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton. Arizona Center for Occupation Safety and Health, U of A Helath Sciences Center Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Annual Surgical Conference - Endocrine Surgery Plus
December 12 & 13, 1980. The Pointe Resort, Phoenix. Sponsor: American College of Surgeons, Arizona Chapter. Contact: F. Darwin Zahn, M.D., 333 E. Virginia, Ste 201, Phoenix, AZ 85004.

9th Annual Seminar - Bone & Joint Diseases II
December 12,13 & 14, 1980. Camelback Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: Alex Newman, M.D., Program Dir., Dept. of Radiology, MCGH, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 8 hours of category 1 credit, Bone & Joint Disease Workshops on the 12 and 14 hours for the seminar.

Surgery
December 13, 1980. Arizona Health Sciences Center, Tucson. Sponsor: College of Medicine. Contact: Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724, Approved for 6 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
December 19-20, 1980. Arizona Medical Association Offices. Sponsor: A.C.E.P., Arizona Affiliate- Amer. Heart Assn., & ArMA. Contact: Carol Turner, Arizona Medical Association, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

1981
JANUARY

Eight Annual Symposium
Barrow Neurological Institute of St. Joseph's Hospital, & Medical Center - Controversies in Neurology and Neurosurgery
January 8-10, 1980. La Posada Resort, Scottsdale, Arizona. Sponsor: St. Joseph's Hosptial and Medical Center, and the Barrow Neurological Foundation. Contact: Richard A. Thompson, M.D., Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Clinical Recognition and Management of Heart Disease 1981
Twenty-Fourth Annual Scientific Session of the American Heart Association, Arizona Affiliate
January 21-24, 1981. University of Arizona College of Medicine, Main Auditorium, Tucson, AZ. Sponsor: University of Arizona College of Medicine, American College of Physicians and the Arizona Heart Association. Contact: Office of CME and Outreach U. of A. Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

FEBRUARY

Seventh Annual Frontiers in Ophthalmology
February 12-14, 1981. The Arizona Biltmore Resort. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator, St. Luke's Hospital. 525 N. 18th St., Phoenix, Arizona (602) 258-7373, Ext. 311. Approved for 15 hours of category 1 credit.

Third Annual Practical Counseling Technique Workshop for the Primary Physician
February 14-21, 1981. Lion Square Lodge, Vail, Colorado. Sponsors: Institute for Human Relationship and Development and Colorado Academy of Family Physicians. Contact: John C. Faul, M.D., 5290 E. Yale Circle, Ste 200, Denver, Co. 80222.

Third Annual International Catarct Surgery Symposium
February 16-18, 1981. La Posada Resort Hotel, Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

Ninth Annual Taos Lung Disease Symposium
February 20-22, 1981. Holiday Inn, Taos, New Mexico. Sponsor: New Mexico Chapter of the American Thoracic Society. Contact: New Mexico Chapter of the American Thoracic Society, 216 Truman, NE, Albuquerque, New Mexico 87108.

Fourth Annual Mid-Winter Symposium in OB/GYN
February 25-27, 1981. Scottsdale Hilton Hotel, Scottsdale, Arizona. Sponsor: Maricopa County General Hospital, Dept. of OB/GYN, and Phoenix OB/GYN Society. Contact: Secretary, Phoenix OB/GYN Society, 333 E. Virginia, Ste 222, Phoenix, AZ 85004. (602) 257-9218.

MARCH

Ski CME Program
March 21-26, 1981. Vail, Colorado. Sponsor: Desert Samaritan Hospital. Contact: L. A. Rosati, M.D., CME Committee, Desert Samaritan Hospital, 1400 South Dobson Road, Mesa, Arizona. Approved for 16 hours of category 1 credit.

Crisis and Emergency Pediatrics
March 5-8, 1981. Doubletree Inn, Tucson, AZ. Sponsor: U of A College of Medicine, and Department of Pediatrics. Contact: Hugh C. Thompson, M.D., Pediatrics, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 17 hours of Category 1 credit.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

LYMAN A. BREWER, III INTERNATIONAL SURGICAL SOCIETY
6TH ANNUAL SCIENTIFIC SYMPOSIUM

on

'CONTROVERSIES IN THE MANAGEMENT OF CORONARY HEART DISEASE"

In Association With White Memorial Medical Center — Loma Linda University School of Medicine

Wednesday, December 3, 1980. 8:00 A.M. to 4:30 P.M. . .and Thursday, December 4, 1980. 8:00 to 4:30 P.M.

The Los Angeles Hilton Hotel

GUEST FACULTY

DAVID H. BLANKENHORN, M.D., *Professor of Medicine, University of Southern California School of Medicine.*

LYMAN A. BREWER, III, M.D., *Distinguished Physician, U.S. Veterans Administration, Jerry L. Pettis Memorial Veterans Hospital.*

B. GREG BROWN, M.D., Ph.D., *Associate Professor of Medicine, University of California, Los Angeles, School of Medicine.*

MORTIMER J. BUCKLEY, M.D., *Professor of Surgery, Harvard University Medical School, Boston, Massachusetts.*

FREDERICK A. HEUPLER, M.D., *Department of Cardiology, Cleveland Clinic Foundation, Cleveland, Ohio.*

WILLIAM P. LONGMIRE, JR., M.D., *Professor of Surgery, University of California, Los Angeles, School of Medicine.*

RICHARD K. MYLER, M.D., *Associate Clinical Professor of Medicine, University of California, San Francisco, School of Medicine.*

MICHAEL P. STERN, M.D., *Associate Professor of Medicine, University of Texas Health Science Center, San Antonio, Texas.*

QUENTIN R. STILES, M.D., *Associate Professor of Surgery, University of Southern California School of Medicine.*

EDWARD STINSON, M.D., *Thelma & Henry Doelger Professor of Cardiovascular Surgery, Stanford University School of Medicine, Stanford.*

RAELENE L. KINLOUGH-RATHBONE, M.D., Ph.D., *Associate Professor of Pathology, McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada.*

FLOYD D. LOOP, M.D., *Chairman, Department of Thoracic and Cardiovascular Surgery, Cleveland Clinic Foundation, Cleveland, Ohio.*

COURSE DESCRIPTION

The 6th annual scientific symposium will present current controversies in the prevention, diagnosis and management of coronary heart disease. A panel of international active clinical investigators will discuss avant garde concepts and clinical findings regarding progress in the prevention and treatment of this disease entity. The course is especially of interest to: the cardiovascular and vascular surgeon, general surgeon, cardiologist, angiography internist, family practice physician, cardiovascular and operating room nurse and perfusionist.

CREDIT

PHYSICIANS: This program offering is approved for 16 hours Category 1 credit toward the California Medical Association Certificate in Continuing Medical Education, The American Medical Association Physician Recognition Award, and the American Academy of Family Physicians.

NURSES: Approved for 16 contact hours of continuing education. Provider No. 00404, BRN State of California.

AMERICAN SOCIETY OF EXTRACORPOREAL TECHNOLOGY:

INFORMATION

REGISTRATION FEES: (Includes luncheons, published symposium proceedings, and refreshment breaks) Physicians $175; Residents, Interns, Nurses, and Others $50. Medical and Nursing Students: Free admission to lectures with letter from Dean.

PLEASE MAKE CHECK FOR APPROPRIATE AMOUNT PAYABLE TO: LYMAN A. BREWER III INTERNATIONAL SURGICAL SOCIETY

MAIL TO: Secretary-Treasurer, LYMAN A. BREWER III INTERNATIONAL SURGICAL SOCIETY,
1720 Brooklyn Avenue, Box 137, Los Angeles, California 90033

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.
Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.
A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.
Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.
Neurosurgical Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third and 5th, Eighth Floor Conference Room.
Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.
Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists
Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.
Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.
Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.
Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.
Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.
Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.
Tumor Board
Last Thursday of the month, 12 Noon, Educ. Center Classroom I & II, May, July, Sept, & Nov.
Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II. Contact: Brian Updegraff, M.D.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.
Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.
CME Programs
Weekly Tuesday, 7 p.m., Cafeteria-West End.
Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.
Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

DOCTORS HOSPITAL

1747 East Thomas Road, Phoenix, Arizona 86016 Contact: Medical Staff Secretary for additional information.

Physicians Continuing Educaiton Program
1st Thursday, 12:30 p.m., Classrooms.

ELDORADO HOSPITAL TUCSON (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712 Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.
Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D Approved for category 1 credit.
Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Con Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 A.M., Contact: Pau Bergeson, M.D.
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm
PM & R Grand Rounds
2st. & 4rd. Wed., 12:00-1:00 P.M., R-2 Conf Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151 Radiology Conf. Rm.
Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr Ben Ora
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.

Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.
Attending's Hour
5th Monday, 12:30 P.M., Contact: J. Goldberger, M.D.
Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman
Ophthalmology Conference
1st Monday, 12:30 P.M., Contact: Bruce Gillers, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.
Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: B. Phibbs, M.D.
Clinical Pharmacology Conference
5th Tuesday, 12:30 P.M., Contact: M. Katz, Clinical Pharm.
12Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact J. Laguna, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: M. Baker, M.D.
Medical Surgical Grand Rounds
2nd Wed., 12:30 P.M., Contact: Chief Medical Resident
Medical Conference
1st Wed, 12:30 P.M., Contact: Chief Medical Resident
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: B. Phibbs, M.D.
Endocrine Conference
2nd Thursday, 12:30 P.M., Contact E. Munjack, M.D.
GI Conference
4th Thursday, 12:30 P.M., Contact: C. Kanun, M.D.
Mortality & Morbidity Conference
3rd. Thursday, 12:30 P.M., Contact: E. Munjack, M.D.
OB/GYN Dept. Meeting
2nd Thursday, 2 P.M., Contact J. Wurzel, M.D.
OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: J Wurzel, M.D.
OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 P.M., Contact: J. Wurzel, M.D.
Clinical Pathology
5th Thursday, 12:30 P.M., Contact: J. Fleishman, M.D.
Renal Conference
1st Friday, 12:30 P.M., Contact: T. Pence, M.D.
Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D.
Rheumatology Conference
3rd Friday, 12:30 P.M., Cntact J. Goldberger, M.D.
Chest Conference
5th Friday, 12:30 P.M., Contct: D. Engelsberg, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008
Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

Clinical Conferences
2nd & 4th Wednesday, 12-1 p.m., Medical Staff Lounge

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 85008. Contact: Leonard Tamsky, M.D.
Medicine Conference
Daily 12-1 P.M., Contact: S. Schaffner, M.D.
Chest Surgery Conference
Weekly, Mondays, 1:30 P.M., Santa Cruz Rm.
Hematology Conference
2nd Tuesday, 1:00 P.M., Contact: Walt Koppenbrink, M.D.
OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Room
Obstetrical Problem Conference
Weekly Tuesday, 7:30 A.M., Yavapai Rm.
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.,Contact: Robert Ganelin, M.D.
Urology Discharge Planning Conference
Weekly, 11:30 A.M., Station 42
Hand Surgery Conference
Weekly Wed, A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly, Wed. 7:30 A.M., Yavapai Rm.
Clinical Psychiatric Conference
Weekly, Wed., 11-12 P.M. Mental Health Annex, Rm. 1346.
Surgery Conference
Weekly Wed., 7-8 A.M., Surgical Dept.
Current Concepts in Medicine & Surgery
1st Thursday, 1 p.m., Dr. Hospital Class Rm., Contact: Dr. Tamsky.
Cardiology Conference
Weekly Thurs., 2 P.M., Santa Cruz Rm.
OB/GYN Resident Conference
Weekly Thurs., 12 P.M., Yavapai Rm.
GYN Endocrine Seminar
1st & 3rd Friday, 12:30 P.M., Sanata Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & HEALTH CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.
Medical & Surgical Conference
Weekly Monday, Wed. and Friday, 12 Noon
OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conf. Rm.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for Category 1 credit.
Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Contact: D. Randall, M.D.
Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 A.M.
Otolaryngology Grand Rounds
4th Wednesday, 4-5 p.m., Conference Rm. A, Contact: N. Wendell Tood, M.D.
Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 A.M., Conf. Rm. A, Contact: Drs. L. Fairbanks & E.Y. Hooper.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for Category 1 credit.
Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Clinical Conferences
Weekly Tuesday, 12 Noon, Kiva Conference Room
Tumor Board Conference
Weekly Friday, 12 P.M., Kiva Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.
Family Practice Conference
1st Monday, 12:30 P.M., Doctors' Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiolpulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs, 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical—Surgical Rdiological Conf.
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.

Genetics Conference
Weekly Monday, 12:30 P.M., Pediatric Department

Pediatric Rounds
Weekly Monday, Wed., & Friday, 10:30 A.M., Pediatric Department

Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.

ECG. Conference
Weekly Tuesday, 12:30 P.M., Pediatric Department

Medical Grand Rounds
Weekly Wednesday, 8 A.M., 1st Floor Conf. Room

Visiting Professor Formal Presentation
Weekly Thursday, 8 A.M., PIMC

Visiting Professor Informal Rounds
Weekly Thursday, 9:30 A.M., 1st Floor Conf. Room

Visiting Professor Formal Presentation
Weekly Thursday, 12:30 P.M., PIMC.

Nephrology Conference
Weekly Friday, 12:30 P.M., Pediatric Department

ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N. Wilmot Road, Tucson, AZ. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.

Family Practice Department Meeting
3rd Tuesday, 12 P.M., Contact Wm. Moteforte

Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 P.M., Contact: Kim Sowards.

Current Concepts In Medicine
Weekly Tuesday, 12 P.M., Auditorium.

Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 P.M., Contact: S. Salmon, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.

Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday

Emergency Medicine Lectures
Weekly Thursday, 8 A.M., Century Room A

Mental Health Update
1st Friday, 11:30-1:00 P.M., Century Room A

Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.

Cardiac Conference
Weekly Monday, 12:15 P.M., Auditorium

Surgery Conference
1st Tuesday, 12:15 P.M., Auditorium

Emergency Medicine Conference
1st Wednesday, 12:15 P.M., Auditorium

Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 P.M., Auditorium

Psychiatry Conference
3rd Thursday, 7 A.M., Auditorium

Combined Medical-General Practice Conference
1st Friday, 12:15 P.M., Auditorium

Toxicology Grand Rounds, Auditorium
2nd Friday, 7:30 A.M., Auditorium

Ophthalmology Conference
1st Saturday, 8:30 A.M., Auditorium.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.

Cardiology Conference
Weekly Monday, except last, 12 Noon, Contact: J. Fernadez, M.D.

Dermatology Conference
4th Monday, 5:00 P.M., Contact: R. Miller, M.D.

Endocrinology Conference
Last Monday, 12 P.M., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.

Chest Conference
3rd Tuesday, 12 Noon, Contact: William Nevin, M.D.

Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.

Infectious Disease Conference
2nd Tuesday, 12 Noon, Contact: B. Friedman, M.D.

Nephrology Conference
1st Tuesday, 12 Noon, Contact: Dr. Seltzer.

Orthopedic Conference
1st Tuesday, 8:00 P.M., Contact: Jay Katz, M.D.

Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 P.M., Contact: Dr. Lightner.

Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.

Clinical Pathology Conference
Last Wednesday, 8:00 A.M., Contact: Dr. Fuchs.

Family Practice Meeting
2nd Wednesday, 12:30 P.M., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.

Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.

Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buschsbaum, M.D.

Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.

Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.

Tumor Conference
Weekly Thursday, 12 Noon, Contact: Dr. Hastings.

G—I Conference
Weekly Friday, 12 Noon, Contact: C. Krone, M.D.

Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.

OBGYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.

OBGYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.

Surgical Department Confernece
Weekly Saturday, 7:30 A.M., Contact: Dr. Hastings, TMC.

Surgical Grand Rounds
Weekly Saturday, 9:00 A.M., UAHSC-Room 5403, Contact: Dr. Norton.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.

Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.

Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403

Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.

Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact: Drs. Otto & Zehngut.

Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 P.M., HSC Auditorium, Contact: Cancer Center

Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.

Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.

Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn

Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore

Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.

Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.

Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch

Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson

GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus

GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter

Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning

Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Room 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC,

FRONTIERS IN OPHTHALMOLOGY

1981

The seventh annual **Frontiers in Ophthalmology** seminar will explore the latest research and techniques in significant areas of ophthalmologic care. A distinguished faculty has been selected to conduct the seminar sessions.

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For additional information, write or call:
Christine Campbell, Seminar Coordinator/St. Luke's Hospital Medical Center
525 N. 18th Street, Phoenix, Arizona 85006/(602) 258-7373 ext 311

Contact: Dr. J. Smith.

Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn

Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern

Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.

Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.

Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford

Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room

Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross

Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross

Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.

Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.

Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley

Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.

Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.

Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson

Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones

Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson

Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655

Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich

Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.

Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.

Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.

General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

PHOENIX VETERANS ADMINISTRATION MEDICAL CENTER

7th Street and Indian School Road, Phoenix, AZ 85012. Contact: Alfred Heibrumn, M.D. Approved for Category 1 credit.

GI X-Ray Clinical Correlation Conf.
1st & 3rd Monday, 1 P.M., Rm. C435, Contact: Drs. Sanowski, & Grobe.

Medical/Surgical GI Conference
1st & 3 rd.Mondays, 3 P.M., Rm. 3134, Contact: Dr. Kozarek, Ext 413. Dr. Mertz, Ext 493.

Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 P.M., RM. 3134, Contact: Dr. Mertz, Ext 493.

GI Pathology Conf.
2nd Monday, 1 P.M., Path Conf. Rm, Contact: Drs. Scowloft & Lenard.

Vascular Conference
2nd Monday, 3 P.M., Room 3134, Dr. Cintora, Ext 419

Surgery/Pathology Conference & Tissue Review
1st & 3rd Tuesday, 4 P.M., Room 2128, Contact: Dr. Landard

Surgery Mortality/Morbidity Conference
4th Tuesday, 4 P.M., Room 3134, Contact: Dr. G. Mertz.

GI Grand Rounds
Weekly Tuesday, 1 P.M., Contact: Drs. Sanowski, Kozarek & Partyka

Cancer Symposium
Weekly Wed., 1 P.M., T-5, Contact: Dr. Byrne, Ext. 426.

Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 P.M., Contact: Dr. Rohwedder, Ext. 388.

Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 P.M., Rm 3134. Contact: Dr. Mertz, Ext. 493.

Medical Surgical Chest Conference
1st & 3rd Thursday, Contact: Dr. Rohwedder or Dr. Gill.

Cardiology Conference
2nd Thursday, 1 P.M., Rm. T-5.

Medical Service Grand Rounds
Weekly Friday, 11 A.M., Contact: Dr. Zeller

Surgical Journal Review & Videotape Session
Weekly 1 P.M., Room 3134.

PRESCOTT VETERANS ADMINISTRATION HOSPTIAL MEDICAL CENTER

Prescott, Arizona 86313. Contacts listed below. Approved for category 1 credit.

Medical Lectures
1st & 2nd Thursday & Weekly,Fridays, 1:30 P.M., Bldg. 28. Contact: J. W. Fitzharris, D.O.

GI Rounds
Third Thursday, 10 A.M.-2:30 P.M., Contact: J. W. Fitzharris, D.O.

Grand Rounds
Weekly Thursdays, 11 A.M., Bldg. 107. Contact: J. W. Fitzharris, D.O.

TUCSON VETERANS ADMINISTRATION HOSPITAL & MEDICAL CENTER (U of A Tucson)

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2 P.M., Contact: Dr. Young

Medical Grand Rounds
Weekly Wed., 12-1:00 P.M., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

Grand Rounds
Weekly, Thursday, 11 A.M., Bldg. 107, Contact J. Fitzharris, D.O.

Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon.

Neurology Grand Rounds
Weekly Friday, 12 P.M., Contact: Dr. Sibley.

YUMA REGIONAL MEDICAL CENTER (U of A, Tucson/ArMA)

2400 Avenue A., Yuma Az 85364. Contact:Alan Winfield, M.D. Approved for category 1 credit.

Radiology Conference
1st Tuesday, 7:00 A.M.

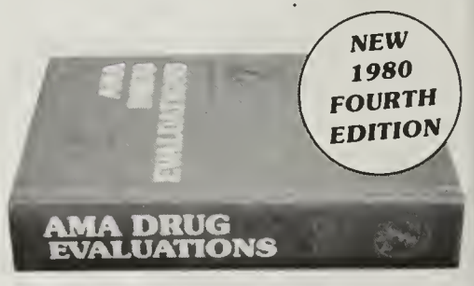
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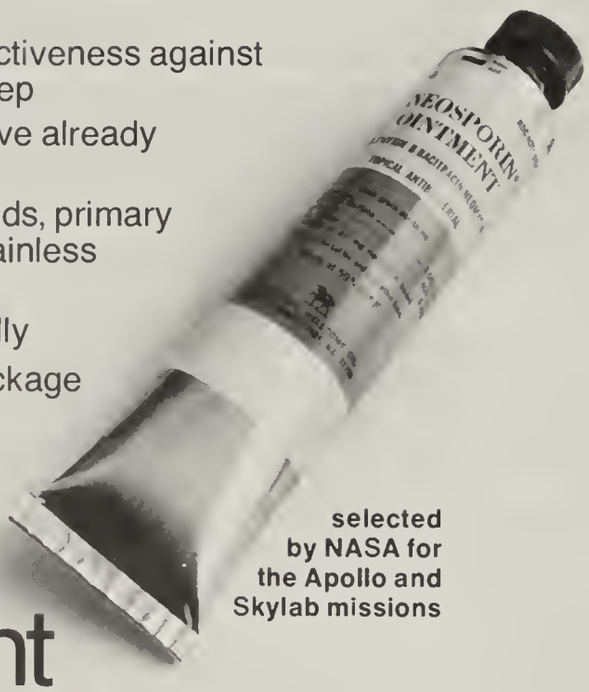
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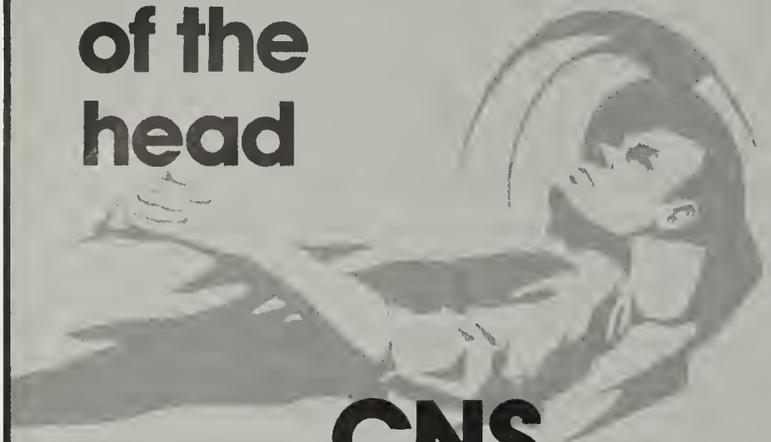
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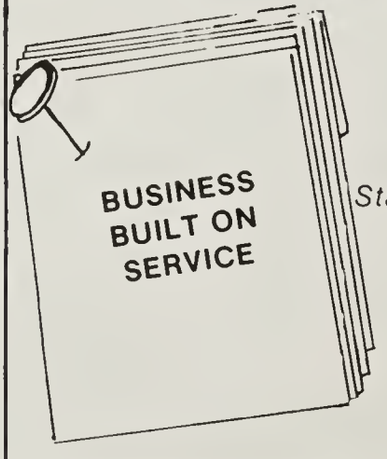
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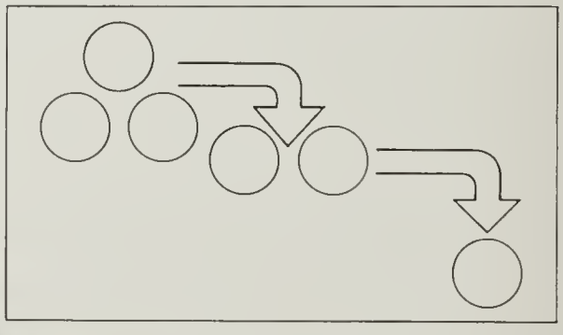
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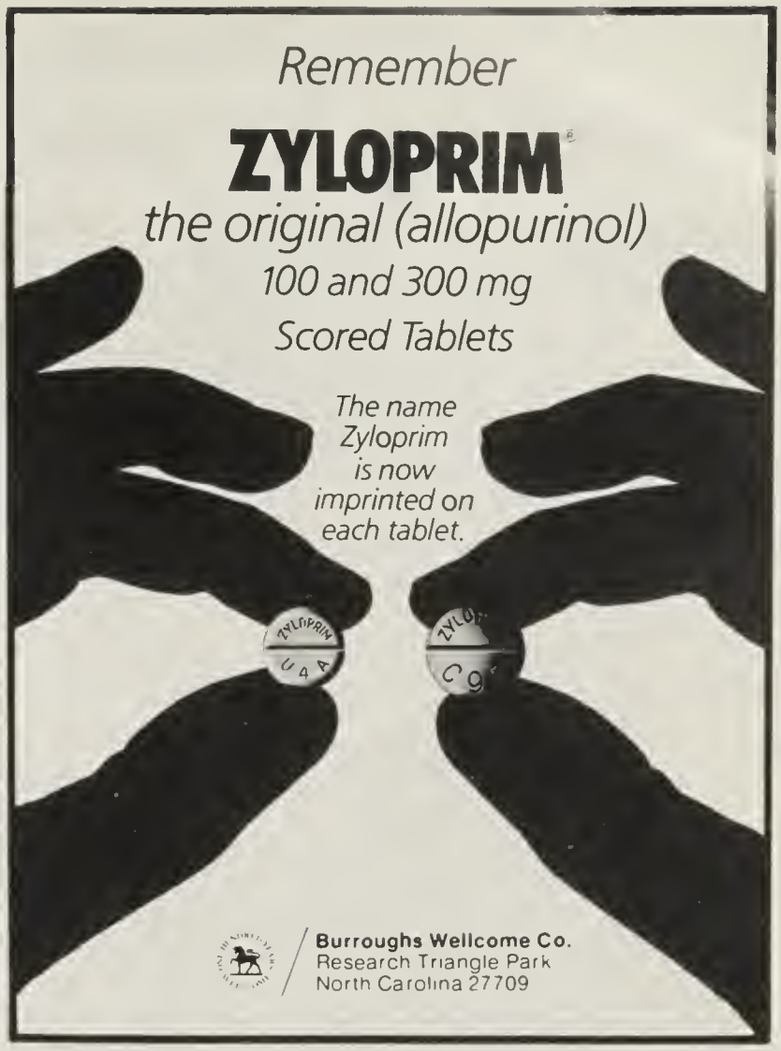
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*Trademark

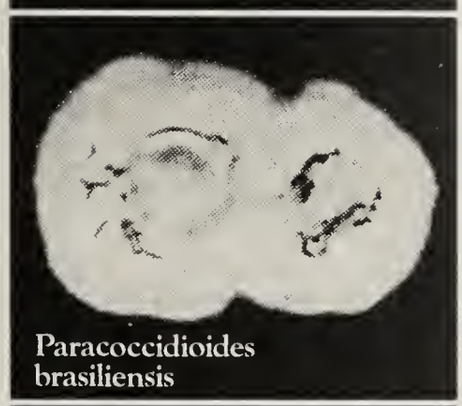
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Candida albicans



Coccidioides immitis



Paracoccidioides brasiliensis



Cryptococcus neoformans

Highly effective, improvement often seen within 1-3 weeks

Patients who do not respond or develop resistance to amphotericin B have responded to MONISTAT* i.v.

Resistance does not develop

In vitro resistance of clinical isolates has not been reported during clinical trials.

Generally well tolerated by severely debilitated patients

Patients with cancer, those receiving immunosuppressive agents, and patients with serious underlying conditions, such as diabetes mellitus or renal failure.

Photos courtesy of Dubos, R.J., Bacterial and Mycotic Infections of Man, J.B. Lippincott Company, 1965 and Burrows, W., Textbook of Microbiology, W.B. Saunders Company, 19th edition.

Please turn page for Prescribing Information.



An important advance in the treatment of systemic mycoses

Monistat i.v.

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(miconazole)

broad-spectrum antifungal agent

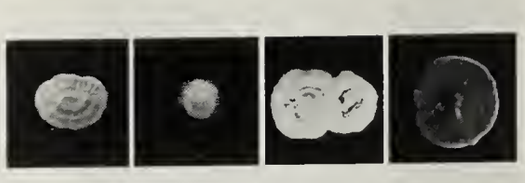
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JPI-024

Broad-spectrum antifungal agent

Monistat i.v.™



(miconazole)

for intravenous infusion

DESCRIPTION: MONISTAT i.v. (miconazole), 1- [2- (2,4-dichlorophenyl)- 2- [(2,4-dichlorophenyl) methoxyl] ethyl]-1H-imidazole, is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v. is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in-vitro antifungal activity of MONISTAT i.v. is very broad. Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis, Candida albicans, Cryptococcus neoformans, and Paracoccidioides brasiliensis.*

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1µg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MIC's should be determined.

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v. may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v. therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported. If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle, Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued.

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported. In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated.

DOSAGE AND ADMINISTRATION:

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended:

Organism	Dosage Range *	Duration of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to >20
Cryptococcosis:	1200 to 2400 mg per day	3 to >12
Candidiasis:	600 to 1800 mg per day	1 to >20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to >16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children. A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium.

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New Brunswick, N.J. 08903

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because so much remains to be done

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Please send me more information about the use of MONISTAT i.v.™ (miconazole) in systemic candidiasis and other systemic mycoses.

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November 1980

Where were they in 1976?

"Where were you in '76?" Be ready with that question when approached by a commercial insurance carrier bent on selling a malpractice policy.

We remember well. The Arizona medical community was close to paralysis when the commercial insurance companies boosted premiums for malpractice coverage astronomically in 1975-76. One company went "belly-up", and another major carrier suddenly pulled out of the malpractice insurance business in Arizona altogether.

MICA was created by physicians for physicians to fill the void. Since then MICA has developed into a strong, stable, and efficient provider of malpractice coverage.

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Now, as of October 1, 1980, the special statutory requirements established by the legislature during the malpractice crisis expired. We expect the climate thus created with regard to physicians' liability insurance to rekindle the interest of commercial carriers in the Arizona market.

Should this occur, there are basic questions which need answering.

MOTIVE What is the company's primary motive for writing policies in Arizona? Is it to benefit physicians and their patients, or is the primary concern stockholder profits?

COST EFFICIENCY What is the company's expense ratio —or, how much of the premium dollar will be used to pay agents' commissions and administrative costs, leaving a diminished balance for the payment of claims?

STABILITY Will there be a repetition of 1976? Is there any assurance that a company will not pull out if it experiences a bad year or two?

RATES How do rates, especially first-year rates, compare? Initial rates may be "loss leader" rates. A study of the company's performance in other states may indicate rapid rate escalations in subsequent years.

Surely, there's a lesson to be learned from past experience!

MICA will continue to provide Arizona physicians with current pertinent information about professional liability insurance in future issues of this publication.

And, MICA will continue to challenge commercial carriers:
"Where were you in 1976?"

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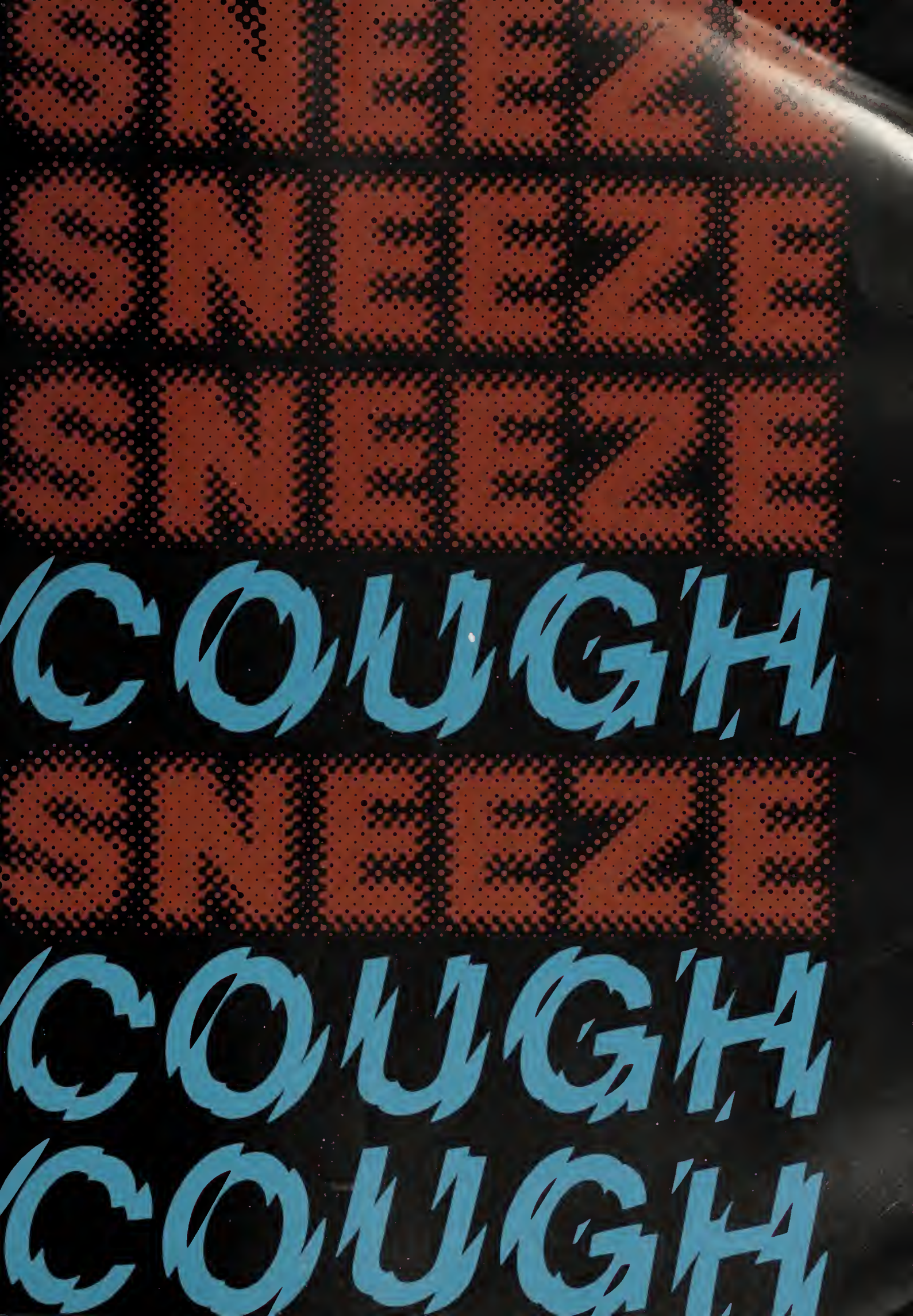
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Chlorpheniramine Maleate	8 mg
Hyoscyamine Sulfate	0.19 mg
Atropine Sulfate	0.04 mg
Scopolamine Hydrobromide	0.01 mg

Ru-Tuss Tablets act continuously for 10 to 12 hours.

- Vasoconstrictor, antihistaminic actions
- Rapid and prolonged relief of nasal and sinus congestion
- Convenient b.i.d. dosage

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Codeine Phosphate (WARNING: MAY BE HABIT FORMING)	65.8 mg
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Phenylpropanolamine Hydrochloride	20 mg
Pheniramine Maleate	20 mg
Pyrilamine Maleate	20 mg
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Alcohol	5%

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- Rapid relief of upper respiratory congestion and cough
- Good tasting

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RU-TUSS® TABLETS

DESCRIPTION
Each prolonged action tablet contains:

Phenylephrine Hydrochloride	25 mg
Phenylpropanolamine Hydrochloride	50 mg
Chlorpheniramine Maleate	8 mg
Hyoscyamine Sulfate	0.19 mg
Atropine Sulfate	0.04 mg
Scopolamine Hydrobromide	0.01 mg

Ru-Tuss Tablets act continuously for 10 to 12 hours
Ru-Tuss Tablets are an oral antihistaminic, nasal decongestant and anti-secretory preparation.
INDICATIONS AND USAGE Ru-Tuss Tablets provide relief of the symptoms resulting from irritation of sinus, nasal and upper respiratory tract tissues. Phenylephrine and phenylpropanolamine combine to exert a vasoconstrictive and decongestive action while chlorpheniramine maleate decreases the symptoms of watering eyes, post nasal drip and sneezing which may be associated with an allergic-like response. The belladonna alkaloids, hyoscyamine, atropine and scopolamine further augment the anti-secretory activity of Ru-Tuss Tablets.
CONTRAINDICATIONS Hypersensitivity to antihistamines or sympathomimetics. Ru-Tuss Tablets are contraindicated in children under 12 years of age and in patients with glaucoma, bronchial asthma and women who are pregnant. Concomitant use of MAO inhibitors is contraindicated.
WARNINGS Ru-Tuss Tablets may cause drowsiness. Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers.
PRECAUTIONS Ru-Tuss Tablets contain belladonna alkaloids, and must be administered with care to those patients with glaucoma, or urinary bladder neck obstruction. Caution should be exercised when Ru-Tuss Tablets are given to patients with hypertension, cardiac or peripheral vascular disease or hyperthyroidism. Patients should avoid driving a motor vehicle or operating dangerous machinery (See Warnings).
OVERDOSAGE Since the action of sustained release products may continue for as long as 12 hours, treatment of overdoses directed at reversing the effects of the drug and supporting the patient should be maintained for at least that length of time. Saline cathartics are useful for hastening evacuation of unreleased medication. In children and infants, antihistamine overdosage may produce convulsions and death.
ADVERSE REACTIONS Hypersensitivity reactions such as rash, urticaria, leukopenia, agranulocytosis, and thrombocytopenia may occur. Other adverse reactions to Ru-Tuss Tablets may be drowsiness, lassitude, giddiness, dryness of the mucous membranes, tightness of the chest, thickening of bronchial secretions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hypertension, faintness, dizziness, tinnitus, headache, incoordination, visual disturbances, mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constipation, epigastric distress, hyperirritability, nervousness, dizziness and insomnia. Large overdoses may cause tachypnea, delirium, fever, stupor, coma and respiratory failure.
DOSAGE AND ADMINISTRATION Adults and children over 12 years of age, one tablet morning and evening. Not recommended for children under 12 years of age. Tablets are to be swallowed whole.
HOW SUPPLIED:

Bottles of 100 Tablets	NDC 0524-0058-01
Bottles of 500 Tablets	NDC 0524-0058-05

Federal law prohibits dispensing without prescription

COUGH

RU-TUSS® EXPECTORANT

DESCRIPTION
Each fluid ounce of Ru-Tuss Expectorant contains:

Codeine Phosphate	65.8 m
(WARNING: MAY BE HABIT FORMING)	
Phenylephrine Hydrochloride	30 m
Phenylpropanolamine Hydrochloride	20 m
Pheniramine Maleate	20 m
Pyrilamine Maleate	20 m
Ammonium Chloride	200 m
Alcohol	5

Ru-Tuss Expectorant is an oral antitussive, antihistaminic, nasal decongestant an expectorant preparation.
INDICATIONS AND USAGE Ru-Tuss Expectorant is indicated for symptomatic relief upper respiratory congestion associated with pharyngitis, tracheitis, bronchitis, an allergic rhinitis. Also, for the temporary relief of symptoms associated with hay feve allergies, nasal congestion and cough due to the common cold.
CONTRAINDICATIONS Hypersensitivity to antihistamines. Concomitant use of an ant hypertensive or antidepressant drug containing a monoamine oxidase inhibitor i contraindicated.
Ru-Tuss Expectorant is contraindicated in patients with glaucoma, bronchial asthm and in women who are pregnant.
WARNINGS Ru-Tuss Expectorant contains codeine phosphate, therefore, the patie should be warned of the potential that this drug may be habit forming. Ru-Tuss Expe torant may cause drowsiness. Patients should be warned of the possible additive effe caused by taking antihistamines with alcohol, hypnotics, sedatives and tranquilizers.
PRECAUTIONS Patients taking Ru-Tuss Expectorant should avoid driving a motor vehicl or operating dangerous machinery (See Warnings). Caution should be taken with tients having hypertension, diabetes, hyperthyroidism and cardiovascular disease. Caution should also be used in patients with pulmonary, hepatic or renal insufficienc
ADVERSE REACTIONS Ru-Tuss Expectorant may cause drowsiness, lassitude, giddine dryness of mucous membranes, tightness of the chest, thickening of bronchial secre tions, urinary frequency and dysuria, palpitation, tachycardia, hypotension/hyperte sion, faintness, dizziness, tinnitus, headache, incoordination, visual disturbance mydriasis, xerostomia, blurred vision, anorexia, nausea, vomiting, diarrhea, constip tion, epigastric distress, hyperirritability, nervousness and insomnia. Overdoses m cause restlessness, excitation, delirium, tremors, euphoria, metabolic acidosis, stup tachycardia and even convulsions.
DOSAGE AND ADMINISTRATION Adults 1 or 2 teaspoonfuls, orally, every 4 hours, not exceed 10 teaspoonfuls in any 24-hour period.
Children 6 to 12 years of age ½ the adult dose, not to exceed 6 teaspoonfuls in a 24-hour period. Children 2 to 6 years of age ½ teaspoonful every 4 hours, not to exceed 3 teaspoonfuls in any 24-hour period. Children under 2 years of age: Use as directed a physician.
HOW SUPPLIED

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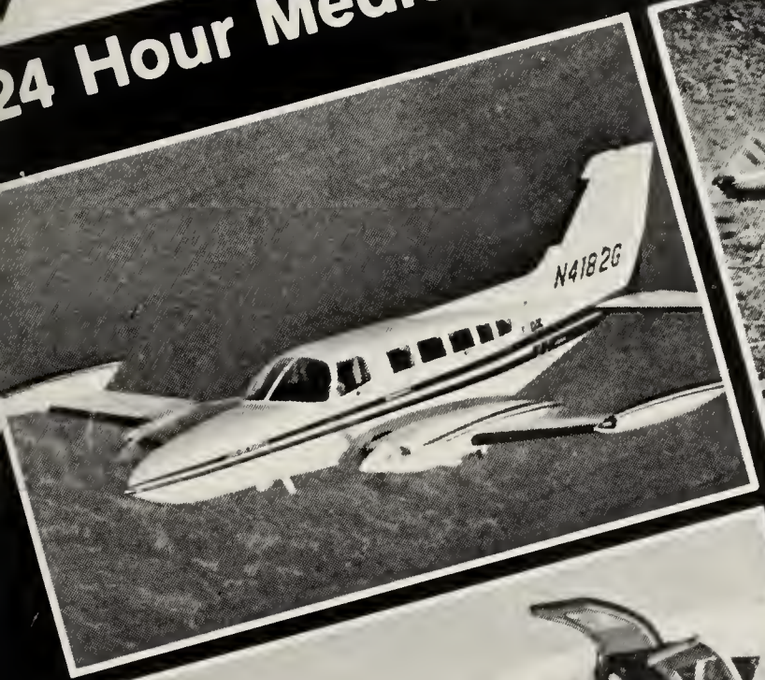
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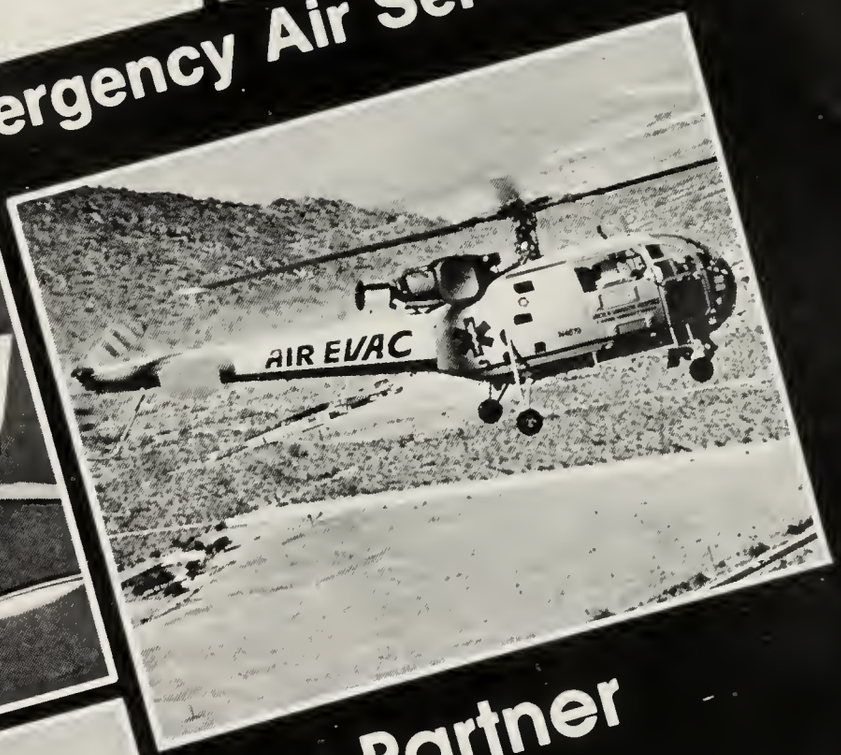
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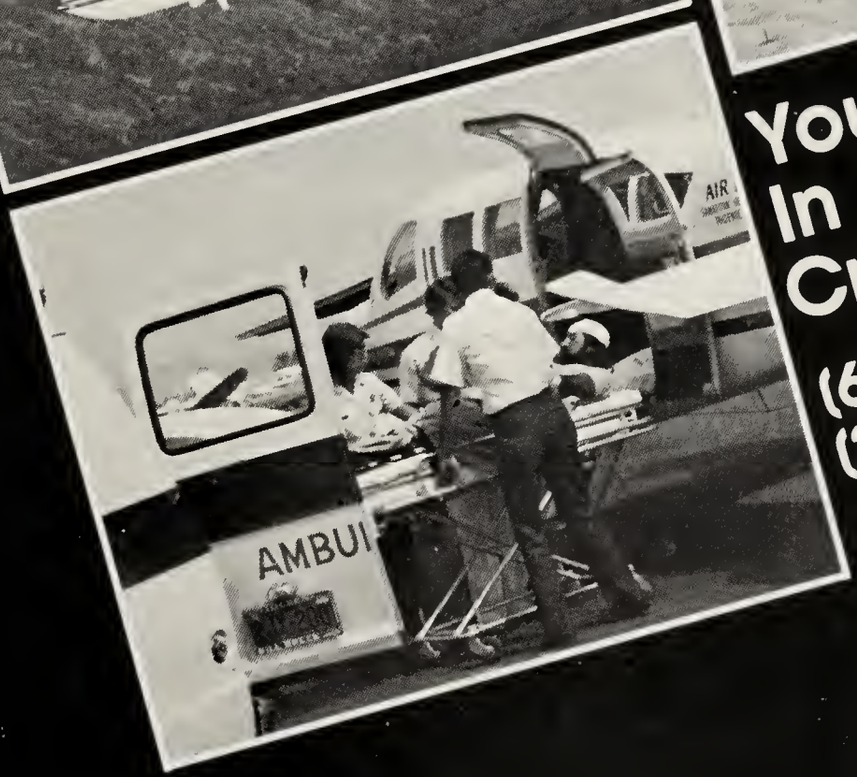
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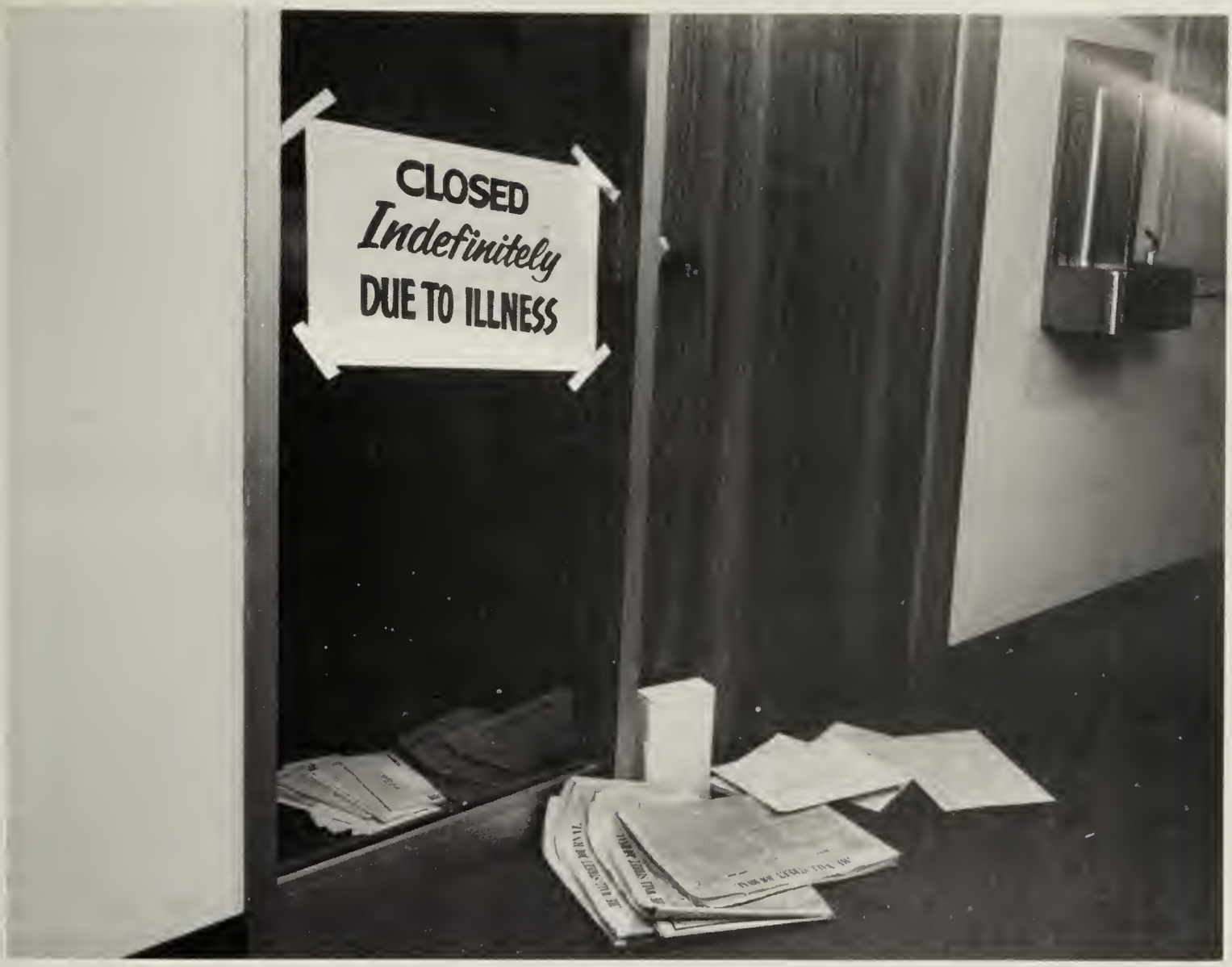
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Cardiology

Recipient Selection For Cardiac Transplantation

Jack G. Copeland, M.D.
Neal W. Salomon, M.D.

Editor:
Kenneth B. Desser, M.D.

In the early days of cardiac transplantation (1967-1969) referral of cadaveric patients, too sick for heart transplantation by current selection criteria, may have been the major cause of high mortality rates. By 1970 it was apparent to those who were still interested that selection criteria for potential recipients were extremely important in determining the outcome of the transplant procedure and were directly related to the ability of transplant surgeons to diagnose and treat rejection. It was clear that if a patient were too sick prior to cardiac transplantation, he might die shortly after the procedure failing to benefit from the effort while consuming a good deal of time and expense prior to death. On the other hand, because of the limitations of cardiac transplantation in prolonging life, it was apparent that if a recipient were not sick enough he might die prematurely, the victim of "successful" heart transplant. In 1972 when survival statistics indicated at 50% chance of one-year survival with heart transplantation, it was stated that a patient would be accepted as a potential recipient only if it was agreed that he faced death within a matter of weeks. Since 1974 the one year survival following heart transplantation has been approximately 65% and the five year survival nearly 50%.[1] A recent summary from Stanford states that "they (potential recipients) should have a poor prognosis for surviving the next 6-12 months and generally are limited by functional class IV congestive failure."[2] It is apparent that with improved survival statistics the selection criteria for potential recipients of heart transplants have broadened slightly. However, until the therapy for cardiac rejection becomes more specific and causes less generalized immunosuppression, it is doubtful that patients who have not reached class IV functional limitation and who do not have a prognosis limited to twelve month survival or less will be considered as transplant candidates.[3]

The ideal candidate for cardiac transplantation is under the age of 50, and functional class IV (cardiac symptoms while at rest.) He should be a patient whose prognosis for survival is less than twelve months on maximal medical therapy, for whom no standard surgical operation has a reasonable chance of success and in whom all noncardiac organ function is normal. It is never possible to pinpoint the prognosis of a given patient to a certain number of months of survival particularly when one has seen the patient only once or twice in consultation. We have found the best indicator of patient prognosis to be the referring physician who has had the opportunity to follow the patient over a period of months and document a progressive decline in function.

These patients most commonly have severely impaired left ventricular function secondary to ischemic or idiopathic cardiomyopathy. Those presenting with the former tend to be predominantly males who are in their forties and fifties. Those pertaining with the latter are generally equally divided between male and female under the age of forty. Both groups generally present with symptoms of a low cardiac output and left ventricular failure a weak pulse, a narrow pulse pressure, pulsus alternans, cool extremities, pale to cyanotic skin, jugular venous distention, hepatomegaly, pulmonary rales, and a greatly enlarged heart revealing a lateral impulse. Also, S3 and S4 gallops and a mitral regurgitant murmur are common findings. Generally these patients at the time of referral for cardiac transplantation have been treated with digitalis, high dose furosemide, afterload reduction, antiarrhythmic agents, and anticoagulants. Nuclear medicine ejection fractions are most often less than 20%.

At present, patients submitted for cardiac transplantation should be under the age of 55 and for optimal results under the age of 50. This statement is based upon survival statistics for different age groups after transplantation. Patients older than 55 have a 15% three year survival as compared to patients under the age of 40 who have a 55% three year survival. This age cut-off, while not absolute, indicates that older patients are more susceptible to the complications of

From: University of Arizona Health Sciences Center, Department of Surgery, Tucson, AZ 85724. Reprint requests to Dr. Copeland, Associate Professor, Surgery Department, Section of Cardiovascular and Thoracic Surgery, U of A Health Sciences Center, Tucson, AZ. 85724

Table 1
Usual Recipient Evaluation:

History and Physical Examination
Routine Laboratory Testing, Blood Type, HLA Type
Pulmonary Function
Skin Testing
Cardiac Catheterization and Angiography
Psychosocial Evaluation by Follow Up Team (Social worker, Psychiatrist)
Financial Evaluation and Counseling

immunosuppression and often succumb to infections which could be tolerated in younger patients.[4]

Any patient who might be considered a potential cardiac recipient should undergo an evaluation similar to that shown in Table 1. The expense of such an evaluation should be avoided if there is present any systemic or non-cardiac organ dysfunction which would limit the recovery or survival of the patient after cardiac transplantation.

Several absolute contraindications for cardiac transplantation have evolved from the experience at Stanford University (see Table 2). These include active infection at the time of consideration for heart transplantation. Such infections can be life threatening on immunosuppressive medication and therefore patients with infections must be first treated and completely cured prior to reconsideration for transplantation. Recent pulmonary infarction has been shown to lead to a considerably increased risk for development of pulmonary abscess in the area of the infarction after treatment with immunosuppressive medications. Therefore, a recent pulmonary infarction is an absolute contraindication for cardiac transplantation and it is best in these patients to treat with Coumadin and wait until scarring has taken place in the lung over a period of 6 weeks to 3 months. Diabetes mellitus, which requires insulin therapy, is a third absolute contraindication for two reasons: 1. Steroids are insulin antagonists and escalate the insulin requirements for diabetes; 2. Diabetics are more prone to infection than the normal population and this predisposition is exacerbated by immunosuppression. Pulmonary vascular resistance of greater than 8 Wood units is a fourth absolute contraindication. Pulmonary vascular resistance is equal to the mean pulmonary artery pressure, minus the pulmonary wedge mean pressure, divided by the cardiac output in liters per minute. It is extremely important that careful determinations be made because a normal heart placed in series with lungs that have severely elevated pulmonary vascular resistance fails almost immediately and patients who have had such elevation in pulmonary vascular resistance have died most frequently within the first twelve hours following transplantation. Finally any mental or psychological condition which would make the patient unable to comply with a difficult medical

Table 2
Primary Selection Criteria:

Irremediable Terminal Cardiac Disease
Age < 55
Non-cardiac Organ Function: Normal or Reversible Dysfunction
Absence of Systemic Illness Which Would Limit Recovery or Survival

Absolute Contraindications:

Active Infection
Recent Pulmonary Infarction
Diabetes Mellitus (Insulin Requiring)
Pulmonary Vascular Resistance, 8 Wood Units
Psychosis or Mental Deficiency Unrelated to Low Cardiac Output
Drug Addiction

regimen over the long term is an absolute contraindication to heart transplanation. Included in this category would be patients with psychosis or mental deficiency and also patients addicted to drugs.

Clearly, there are many other conditions which might contraindicate transplantation. Most common among these is probably chronic obstructive pulmonary disease, particularly chronic bronchitis with sputum production. These patients have little chance of survival after immunosuppression since they are very likely to be afflicted with pneumonia.

Patients who may be potential cardiac recipients should also be screened sociologically and financially. It has been found that patients who have a stable social base with responsive family members or relatives who can lend psychological support as well as physical assistance throughout the post-transplanation period are vital for a good prognosis. Patients who have dealt unsucessfully with other stresses in life are likely to be unsuccessful in dealing with stress of cardiac transplantation. Finally, some form of financial support should be available prior to committing a patient to cardiac transplantation. It must be realized from the outset that the initial cost of cardiac transplantation at the Arizona Health Sciences Center will be in the range of $20-30,000 and that subsequent hospitalizations particularly in the first year after transplantation may be necessary. For this reason it is important that the patient have either good comprehensive health insurance coverage or coverage by Champus, the Veterans' Administration Hosptial, or Medicare. In the absence of health insurance coverage, patients are required by the Arizona Health Sciences Center to provided evidence of assets available for payment of hospital costs of $25,000 in advance of hospitalization. This requirement discriminates against the medically indigent patient and is currently one of the most frustrating shortcomings of our program.

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Editorial Comments

Kenneth B. Desser, M.D.

Cardiac transplantation is here to stay. Arizona is a beneficiary of the intrepid activities of the Stanford University surgical team which persisted through the years of scientific flagellation. It is of interest to note that the trustees of the Massachusetts General Hospital decided against allowing the hospital's cardiac surgical service to begin a limited program of heart transplantation. Their decision was based on three issues: 1) therapeutic efficacy of the procedure, 2) scientific fallout and 3) allocation of costly and limited resourses (*N Eng J Med 302:1087-1088, 1980*). As of 1980 the five year survival following cardiac transplantation is approaching 50%. Insofar as interdisciplinary comparisons are valid, this astounding success rate stands up favorably to data provided by cancer chemotherapeuticians, and those derived from standard surgical procedures utilized for malignant neoplastic processes. Indeed the statistics resemble those of chronic hemodialysis during its incipient stages. Curiously, these other medical therapies do not seem to evoke as much scientific outcry or reservations based on resource allocation. In fact, they are standard procedures accepted as usual within most major tertiary care hospital centers. Medical economists tell us that there must be a limit to what we can do. They coin phrases such as "cost-effectiveness" and expound on the increasing slice of the gross national product pie demanded by medical care. Witness the huge lines for $5.00 junk cinema, consumption figures for junk food, junk music, cigarettes and hard liquor. In the light of such expenditures and their "cost-effectiveness" $25,000 appears to be a bargain for a 50% chance to remain alive for 5 years. Cardiac transplantation is a "Rolls-Royce" surgical procedure. Productive members of society disabled by Class IV congestive heart failure during the prime of their life deserve to travel first-class. When the rejection problem is solved they will be lining up in droves. It is forecast that the problem of the future will be donor availability.

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Emergency Medicine

Electrical Injury

John H. Hughes, M.D., FACS

Editor:
Marcus R. Engelman, M.D.

Acute and chronic injuries related to electrical current cause over 1,2000 deaths a year.[1] Cooper has noted that the death of 300 persons per year in the United States from lightening exceeds those killed by tornados.[2] Adverse effects of electrical current passing through the human body have been described by Pudenz,[3] Hunt, et al,[4] and Artz[5]. Leeming[6] has described protection of the "electrically susceptable patient." Severe acute, adverse effects include burns, cardiac, neurological and muscle injuries.

In order to review our own experience patients presenting with electrical injury, the records of University Hospital in Tucson, Arizona, were reviewed from 1971 through 1979. During that period no patient seen here had been struct by lightening. One person has been seen after lightening injury in 1980. This involved no burn or major injury, but muscle pain and fasiculations in a girl's arm that had been extended out a car window when struck. The abmulatory and inpatient records of all 16 patients who presented with adverse effects of electrical shock during that period have shown diverse circumstances of injury.

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Cases

1. A 61-year-old Mexican/American was seen for progressive weakness three years after an electrical shock in a machine shop. The patient had been confused and restless for five and a half days following initial injury. He had weakness, numbness in the arms, impaired balance, and chest discomfort along with dysarthria and spastic quadriparesis. It was felt that he had delayed spinal cord injuries.

2. A 23-year-old white male was seen following injury from a 440 volt cable grasped in his non-dominant right hand on the day of his admission. There were no systemic injuries documented. The patient was treated with excision and grafting with a good result.

3. A 39-year-old white male made contact with a live wire of a generator which carried 13,000 volts. He was knocked approximately ten feet through the air striking his back and elbow against concrete and collapsing. The patient did not lose consciousness but felt extremely weak following the event. The patient showed no dysrhythmia or laboratory test abnormalities during 24 hours of observation.

4. A 63-year-old white male received an electrical shock while working at a drill press. While standing on a rubber mat, he received the shock accompanied by transient visual loss and a tingling sensation throughout the body followed by pain and weakness in the right arm and shoulder. He was admitted for osbervation, showed no dysrhymia or abnormal systemic signs and was discharged asymptomatic on the following day.

5. A 15-year-old white male received electrical shock while talking on a phone in a laundromat. The phone cord apparently short circuited with the coin changing machine while the patient was in contact with both objects. Following syncope on the way to a hospital the youth exhibited vague personality changes. Neurological evaluation including EEG, brain scan, lumbar puncture, EKG, and laboratory tests were within normal limits.

6. A 33-year-old patient was seen three months following an electrical shock with entrance through the head and exited through both knees. He experienced generalized pain in the low back and anterior chest yet returned to work the day after the injury. Assessment of the patient showed no abnormalities.

7. A 15-year-old white male was seen shortly after he was rendered unconscious following an attempt to jump over an electrical fence. The boy had bare feet on damp ground and apparently was unable to release the electrical wire for approximately five minutes after initial contact. Deep electrical burns were present on both hands but no systemic injury was identified. He did well following excision and primary grafting.

8. A 64-year-old Mexican/American male was seen for injury to the sole of the right foot after stepping on a high voltage electrical line. No systemic injury was noted. The burn was debrided and grafted with good results.

9. A 55-year-old white male was seen for electrical shock after pulling a 220 V. fuse with pile[illegible] He was asymptomatic and unburned on arrival at the h[illegible] He was discharged from the Emergency Departmen[illegible]

10. A 30-year-old white male was seen for back, leg, right hand, and forearm discomfort following contact with a short circuit in a 220 volt fuse box. Lack of muscle atrophy or loss of deep tendon reflex along with occasional inadvertent muscle activity in the right upper extremity, led to the clinical impression that this patient had a hysterical paralysis subsequent to his injury. Following neurological and endocrine evaluation, the patient was referred for psychotherapy.

11. A 19-year-old male was thrown across his room when plugging in an electric light. His fingers were burned and premature ventricular contractions were present along with burning chest pain. He was treated with lidocaine and monitored for three days. Stress test before discharge showed disappearance of premature ventricular contractions.

Five children with electrical injuries were seen during this nine year period. Three of these involved burns of the mouth resulting from chewing on electrical cords.

Discussion

Adverse effects of electrical discharge are most commonly related to burn, neurological, or cardiac injuries. Studies by Hunt, et al,[4] suggest that initially skin provides good resistance to the flow of current which is then diminished after initial tissue injury. With subsequent charring of the tissues involved, the resistance to flow of the current increases once again. Current appears to flow most rapidly through nerve or blood, least rapidly through bone or fat and at a moderate rate through muscle, skin, and tendon. Baxter[7] describes electrical injuries associated with (a) entrance or exit of electrical current (b) arcing produced by current coursing external to the body from the contact point to ground, or (c) from flame burn associated with ignition of clothing or other objects by the current.

Initial prehospital care involves proper extrication of the individual from the source of current without the rescuer becoming involved in the circuit. Cardiopulmonary resuscitation may be lifesaving. Apnea may result from neurological injury or tetanic contraction of the chest musculature. Dysrhythmias commonly include tachycardia and minor ST segment alterations persisting several weeks following electrical injury.[7] Mycoardial infarction following electrical shock is reported infrequently. Kirchner et al[8] have described a young man in whom cardiac rupture occurred after myocardial coagulation necrosis leading to free ventricular perforation.

Nervous system tissue has least resistance to electrical current and also is highly susceptible to thermal injury. Pudenz,[3] Christiansen et al,[6] and Baxter[7] have described many of the acute and delayed neurological sequalae

Table 1
Evaluation of the Victim of Electrical Injury

1.) Diagram the mechanism of injury where possible noting:
 A. Site of energy entry
 B. Site of energy exit
 C. Presence of conductors and insulators (i.e. dry gloves, rubber-soled shoes, metal braces on teeth)
 D. Voltage involved
2.) Review underlying health and risk factors of the victim.
3.) Obtain EKG, or monitor cardiac pattern if suspicious of cardiac injury or dysrhythmia.
4.) Determine at first clinical encounter the neurological status, including documentation of head injury, loss of consciousness, convulsion, visual activity, recent memory, and psychological status.
5.) Document CPK isoenzymes and urine myoglobin determinations where history suggests muscle injury.
6.) Clean and appropriately treat any area of burn including adequate hydration and tetanus prophylaxis.
7.) Use appropriate consultation and plan careful follow-up after initial care is provided.

associated with various forms of electrical injury. Pudenz[3] noted that monophasic direct-couple waves of current are more destructive to tissue of the nervous system than biphasic pulses. This increased effect of direct-coupled waves also applies to greater disruption of the blood-brain barrier and to vasoconstriction. Christensen et al[8] notes a 60% mortality asssociated with hand to hand passage of current compared to 20% mortality with current passing from hand to foot. They relate this to injury to the spinal cord between C_4 and C_8. They note that while immediate injury to the cord is more common, delayed injury may be seen. Baxter[7] considers neurological complications the most common non-lethal injuries following electrical injury. He notes that partial sensory or motor peripheral nerve lesions frequently regenerate. Autonomic nerve injury may lead to causalgia. He notes further that spinal cord lesions are seldom complete, frequently presenting with spastic paresis with little or no sensory deficit. Transient loss of consciousness is common, but recovery is most frequently seen.[10] Late sequellae of electrical injury include convulsions, cataracts, and causalgia.[7]

Most studies of electrical burn have come from large burn units. However, physicians in different disciplines may be called to see occasional cases following initial injury in which burn is only a small part of the patient problem. In most cases complaints will be transient and self-resolving. However, late sequellae are not rare. Table 1 provides a checklist which may aid in assessment of patients with such injuries.

Conclusions

Medical records of University Hospital in Tucson, Arizona from 1971 through 1979 showed 16 patients presenting with electrical shock. None involved lightening. Three involved children chewing electrical wires. One patient has been seen in 1980 after being struck by lightening.

Initial prehospital care may be lifesaving. Guidelines for initial assessment of the victim of electrical injury have been suggested.

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Seminars in Gastroenterology and Liver Disease

Mallory-Weiss Syndrome

Steven R. Kanner, M.D.

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Introduction

A little over 50 years ago a syndrome of fatal gastrointestinal bleeding secondary to gastric mucosal laceration was described by the chiefs of Pathology and Internal Medicine at the Peter Bent Brigham Hospital in Boston. All of their patients were alcoholics who had massive hemorrhage preceded by retching or vomiting. Indeed, until flexible fiberoptic endoscopy was widely used, this syndrome was characterized, as noted above, as a cause for massive gastrointestinal bleeding, generally requiring surgery and associated with a very high mortality. However, with the advent of flexible endoscopy our view of the Mallory-Weiss syndrome has changed.

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Pathophysiology

Mallory-Weiss syndrome is characterized by a longitudinal mucosal tear which generally occurs in the cardia of the stomach. It occurs most commonly, though not exclusively, in patients with hiatus hernia. Physiologically, nausea causes pylorospasm and distention of the stomach and esophagus associated with counter peristalsis, projecting gastric contents toward the upper portion of the stomach and esophagus. Vomiting, in addition, causes the diaphragm to distend and the abdominal muscles to contract simultaneously causing a marked increase in intragastric pressure. It is this combination of events which induces the gastric mucosal tear. Other situations in which there is increased intragastric pressure, either due to sudden descent of the diaphragm or increased abdominal pressure, have also been associated with the Mallory-Weiss syndrome. These have included coughing, straining at stool, hiccuping, status asthmaticus, seizures, gastroscopy, childbirth, heavy lifting, and cardiorespiratory resuscitation.

History and Physical

The patient population with Mallory-Weiss syndrome are 2:1 male:female, varying in age from adolescence to the elderly. While the most common presenting symptom is hematemesis, the classic history of preceding non-bloody emesis or retching can be obtained in only a third of the patients. Occasionally patients will present with other symptoms, including melena, syncope, and rarely, abdominal pain. Many of these patients have a history of heavy alcohol or aspirin use. While Mallory-Weiss syndrome has in the past been characterized as presenting with massive hemorrhage, approximately one half of patients with this syndrome will exhibit no evidence of significant volume loss at the time of admission. At endoscopy a longitudinal mucosal tear is evident, generally occuring below the GE junction (90%). Occasionally more than one mucosal tear will be noted. Approximately one-third of patients will have an additional abnormal finding found at endoscopy, most commonly ulcer, gastritis, esophagitis, or duodenitis. Indeed, second sites will be found to be actively bleeding in approximately 15% of patients. A patient who we recently evaluated for upper gastrointestinal bleeding was found at endoscopy to have both a Mallory-Weiss mucosal tear and an aortoduodenal fistula.

Management

While Mallory-Weiss syndrome has in the past been characterized as associated with massive bleeding, the majority of patients with this syndrome have no or minimal transfusion requirments. Most patients require nothing more than supportive managment, i.e. replacement of fluids and blood. In some centers electrocautery has been used at the time of endoscopy to control bleeding with approximately 50% success. However, this remains an experimental treatment modality. Vasopressin infusion, either intra-arterial or intravenous, has been useful to control bleeding in those

few patients with continued or recurrent bleeding. In those occasional patients who do have bleeding unresponsive to medical therapy, operative therapy, i.e. oversewing the bleeding site, will generally resolve the problem. While mortality in older series was frequently in the range of 50%, mortality presently is judged to be somewhere between 5-10%. This lower mortality is somewhat attributable to the fact that we are identifying this lesion more commonly in patients with less severe bleeding.

Summary

Mallory-Weiss syndrome is an entity characterized by a mucosal tear at or about the gatroesophageal junction associated with hemorrhage varying from minimal to life threatening. With the advent and widespread use of flexible fiberoptic endoscopy we have come to realize that it is one of the more common causes of upper gastrointestinal bleeding. Whereas it had initially been thought to be associated generally with severe life threatening hemorrhage requiring surgery, we now appreciate that it is much more commonly a cause for mild to moderate gastrointestinal hemorrhage for which surgery is not often required. Mallory-Weiss syndrome offers an example of a medical problem whose "scope" has changed during the course of the years, not because of an actual change, but rather because of a better appreciation of the true spectrum of this entity because of new technology.

Medical Genetics

With this issue we initiate a new section on Medical Genetics. The role genetics plays in health care has increased significantly. Many of the genetic disorders are now definable, treatable, and in some cases preventable. It is the intent of this Section to discuss the myriad aspects of genetics in medicine. We are interested in the genetic observations of the readership, either personally or within your practice. Contributions to this section are most welcome.

Editor:
Frederick Hecht, M.D.

The Fragile X Chromosome Mental Retardation and Large Testes

Frederick Hecht, M.D.
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A major new chromosome abnormality accounts for mental retardation in many males and some females. This chromosome abnormality is the fragile X.

The fragile X chromosome has a constriction near the bottom of the long arm, which is fragile and tends to break easily.

Mental Retardation in Males

Since 1938 it has been known that more males than females are retarded.[1] However, the X-linked disorders known to be associated with retardation were far too rare to account for the significant excess (often 30-50%) of males. Now it is clear that the fragile X is a frequent cause of mental retardation in males.

No male with the fragile X has yet been discovered with normal intelligence. All males with the variant X chromosome are retarded, and the range of mental retardation extends from mild to severe. Most cases apparently are moderately retarded.[2] However, we still do not know the complete picture about retardation in males with this disorder.

Mental Retardaton in Females

Females with the fragile X may be intellectually normal or, in some cases, they may suffer intellectual deficit. If affected, females have less retardation than males.

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Schoolgirls functioning in the mildly retarded (I.Q. 55-75) range were recently investigated.[3] Of 128 girls, 56 had physical abnormalities (such as short stature, microcephaly or abnormal facial features). None of these physically abnormal girls had the variant. The remaining 72 girls had no physical abnormalities. Five of these 72 girls (7%) carried the fragile X. From this study, it can be concluded that female carriers contribute to mild mental retardation in girls. Further, it can be concluded that mildly retarded girls with this disorder are physically normal.

Less Retardation in Females

Females with the fragile X may be mildly retarded or they may be normal in intelligence. The reason for this variation in females is thought to relate to the behavior of the X chromosome. In every female cell, only one of the the two X chromosomes is active. Genes on the other X chromosome are largely inactive. (This concept was originally proposed by Mary Lyons and Ernest Beutler and is now proven.)

The process of X-inactivation is random. In some female cells, the X from the father is inactivated and in other cells the converse is true. Fragile X girls with mild retardation may tend to have the normal X they received from their father inactivated, while fragile X girls with normal intelligence may have the abnormal X they received from their mother inactivated. A similar explanation has been invoked to account for why some female carriers of hemophilia bleed, while others do not.

Large Testes

In a study by Turner et al,[2] twenty-four of twenty-five males with the fragile X had large testes. Large testes (macro-orchidism) may thus prove to be a helpful clue to diagnosis.

Testicular measurements can be made with a centimeter ruler, measuring the length and width, or an orchidometer can be employed. (Most orchidometers resemble a set of oval fishing weights to which one compares the size of the testis). In our experience, most boys not like the procedure of having their testes measured, so the procedure should be done gently toward the end of the physical examination.

Other Anomalies in Males with the Fragile X

The picture is not yet clear on other physical anomalies that occur in males. In the original report of the fragile X, microcephaly was observed in one case.[4] Another report[5] described affected males in four families as "essentially phenotypically normal." However, it was noted[2] that there is a "suggestion of features in common." These features include a large forehead, prominent chin, large ears and pale blue irides. The pale irides may, we believe, reflect ascertainment bias, since the authors work in a country where most people are of Anglo-Saxon origin (Australia).

Ethnic Distribution of the Fragile X

Most cases of the fragile X have , in fact, been reported from countries of largely Anglo-Saxon origin. Is this accidental? We think it is.

A black male has now been reported.[6] More reports will be needed, however, to establish whether the disorder affects blacks with appreciable frequency.

In Arizona we have found a very large family [illegible] the fragile X. The mother's ancestors came directly to Mexico from Spain. She has three retarded sons, all with dark eyes.

The overall ethnic distribution of the dysfunction is thus uncertain. Arizona, with its extraordinary ethnic composition, provides an unusual opportunity to learn whether the fragile X is evenly distributed.

Laboratory Studies

Prior to 1976 reports of the fragile X were sparse.[8] If it is so common, why was it not commonly seen? The reason lies in the laboratory. Most chromosome studies have been done on cells, usually peripheral blood lymphocytes, cultured in complete media containing folic acid and thymidine.

It was then shown that the expression of the fragile X is inhibited by folic acid and thymidine.[9,10] Medium 199, which is deficient in folic acid and thymidine, is used to detect the fragile X.

Prenatal Diagnosis

In 1980 a major advance has been made indicating that the fragile X is visible under special circumstances in skin fibroblasts.[11,12] This now makes it potentially possible to demonstrate the fragile X in amniotic fluid cells. Prenatal diagnosis has not yet been reported, but we expect it will be feasible shortly.

Biochemical Mechanism

We believe that, the fragile X is a biochemical disorder. The reasons relate to 1) the very discrete nature of the fragile site on the X chromosome and 2) the fact that expression of the fragile X is inhibited by folic acid and thymidine .

Glover[13] has studied the mechanism by which folic acid and thymidine inhibit the expression of the fragile site on the X. Using 5-fluorodeoxyuridine (FUdR), the fragile site was induced to express itself in the presence of folic acid. This indicates that (since FUdR is a potent inhibitor of thymidylate synthetase) depletion of deoxythymidine monophosphate, available for DNA synthesis, causes expression of the fragile X.

Therefore, it appears that full understanding of the biochemical mechanism of the fragile X is close at hand. We predict this will be the first chromosome abnormality to be fully fathomed on a biochemical level.

Screening

A family with two or more mentally retarded males should be screened for the fragile X. The males may be in different generations, for example, a boy and his maternal uncle. Large testes provide a second clue and should also lead to screening. Finally, any male with unexplained mental retardation should be screened.

Summary

The fragile X chromosome is a major cause of mental retardation in males. Large testes are a clue to the diagnosis. Also, the fragile X can cause learning

problems and mild retardation in females. No clue, analogous to large testes, is yet available in females. Special chromosome screening, using medium deficient in folic acid and thymidine, is needed to detect the fragile X. It is likely that the fragile X chromosome causes retardation through a simple biochemical mechanism. We anticipate that prenatal diagnosis of the fragile X will be made in this decade. Further, we expect that the disorder may be treatable and retardation preventable.

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Neurology

Management of the Chronic Pain Patient

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Pain is one of the most dramatic, complex and universal phenomena. There are numerous definitions—none are truly adequate.

To the person experiencing pain there are many implications: it signifies emotions ranging from fear to anxiety and punishment, even love or loss of love; pain can be a means of communication, of judgment, or a reason for either obtaining or avoiding treatment. Professionally, it is an important area of research. Equally important, it is a major reason for dispensing clinical treatment by health practitioners in a wide range of specialties.

The literature dealing with pain is voluminous. Clinicians and researchers in practically every specialty have contributed significantly. There seems to be one concept that is almost universally agreed upon. Namely, no single discipline can supply the answer to the multiple concerns of pain.

A discourse of this type does not lend itself to the innumerable intricacies of the chronic pain syndrome. For those who have interest, the literature is replete with explanations of the general and theoretical concepts of pain reaction. Numerous authors have written concerning the measurement of pain. Of great

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interest are those contributions on the social and psychological correlates of pain perception. Cultural influence and the effects of various disease processes associated with chronic pain are also broadly dealt with in the literature of the various specialties.

What are the characteristics of chronic pain? Among the chronic pain patterns seen by the general physician are those resulting from Herpes Zoster, Tic Doloreaux and other facial pain, reflex sympathetic dystrophy, myofascial inflammation, acute low back strain, postoperative incisional pain, and even post-fracture pain.

It is now generally agreed, regardless of etiology, that the constellation of symptoms accompanying the patient with chronic pain is characteristic enough to constitute a disease entity called "The Chronic Pain Syndrome." Pain, illness or environmental changes must be present for about six months to establish this condition as an entity and to be labeled a "chronic pain syndrome."

Once established, often regardless of etiology, patients suffering from this illness will frequently have complaints which may be multiple and often inappropriate to existing somatogenic problems. Their history will include multiple physician contacts and many non-productive diagnostic procedures. They may have an altered behavior pattern and some may exhibit depression, anxiety and neuroticism.

The longer any pain remains ineffectively treated, the greater the probability that major interpersonal components will develop in association with the pain. It will become more difficult to provide an effective regime of therapy.

This is particularly so since pain is an acceptable affliction in our society and especially worthy of sympathy. Friends and relatives may act to sustain invalid behaviors by their well-meaning attention.

The chronic pain patient, after multiple physican contacts, after numerous investigative procedures, and even after much therapeutic intervention, (laparotomies, laminectomies or discectomies), is now very susceptible to any approach, even quackery. This is truly unfortunate, although for those with benign disease their only true loss will be an economic one.

As with any medical problem, a proper diagnosis is essential before proceeding with therapy. A true diagnosis is often particularly difficult in these patients because most come with an established diagnosis. This may have been established by exclusion or is uselessly outdated. Even with an established diagnosis it is necessary to discover the cause of the patient's pain problem.

By the time the chronic pain syndrome has been identified, it has already been proven to be resistant to easy management by accepted, conventional medical approaches.

A fairly new concept in therapy for this disease entity is the multidisciplinary pain clinic. Experience has demonstrated this approach may be most effective in a large university setting. However, many practitioners have set out to disprove that viewpoint. In a cooperative effort among representatives of various specialties pain control centers have been established.

Basically, a physician undertaking the primary management of a patient with chronic pain syndrome must decide if he has the resources and is willing to manage this type of patient. Very often these patients will prove to be more difficult for the physician than other types of patients. The physician and his colleagues will become the focus of both patient and family hostilities. Financial return is frequently not adequate because of the long duration of the patient's illness and the reluctance of third party carriers to pay for the variety of necessary services.

In a multidisciplinary pain clinic the workup commences with simultaneous evaluation of all factors which affect the patient—physically, mentally and environmentally. The patient's intimate family must be involved, preferably the spouse. Once the diagnosis has been made, it should be carefully explained to the patient and their family.

If possible, the patient must be granted the opportunity of referring to one physician who will serve as his or her focus of management. The patient must be allowed to rely upon this one physician, who is responsible for their overall care, regardless of the number of additional referrals the patient may experience. That physician acts as a manager of the patient's problems and progress.

If the patient is a chronic abuser of pain medication, this should be approached at once. Not infrequently chronic users of analgesic medication report less pain after these medications are discontinued. It is difficult to manage patients who abuse pain medications on an outpatient basis. If this presents as an obvious problem, they are best treated as inpatients in one of the several acceptable therapeutic programs. When the medication problem has been controlled and the diagnosis made, the patient is now ready for a direct approach to the treatment of their pain problem.

Management of the psychoses, neuroses and personality disorders seen in the chronic pain syndrome requires access to either a clinical psychologist or psychiatrist interested in pain problems. Behavioral factors and pain-related environmental manipulation often respond to the application of learning principles for the modification of pain behavior. Other modalities of treatment in current use are transcutaneous nerve stimulation and biofeedback training. Hypnosis has played an important role in the treatment of some patients suffering chronic pain.

Often various pain syndromes respond to modalities not encompassing the psycho-social sphere. Such pain patterns as trigeminal neuralgia, the acute and post-herpetic syndrome, the acute lumbo-sacral strain frquently are adequately treated by interruption of sensory nerve pathways and possible instillation of a

steroid combination. The large group of sympathetic dystrophies are often responsive to interruption of sympathetic nerve pathways. The chronic, intractable pain of cancer often responds to intrathecal neurolysis or to pituitary adenolysis. It is interesting that, according to Woodford, most cancer patients do not have pain until they are made aware of their disease.

For that group of patients who are not candidates for a definitive corrective procedure and who suffer from a benign process, supportive therapy must be continued. Often that is sufficient treatment to keep patients from addictive medication. This therapy is best supplied by the physician-manager, the psychologist or, on occasion, by an informed social worker.

The treatment of the chronic pain syndrome is a long, involved, often frustrating patient-doctor relationship. It should be entered into with concern and understanding by the the therapist who wishes to devote his energies to this category of patient.

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Oncology

Results of Adjuvant Therapy For Breast Cancer in Phoenix

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The adjuvant use of chemotherapeutic agents has been widely studied in an attempt to improve survival after mastectomy for breast carcinoma.[1] One study, the large controlled Milan trial, employed cyclophosphamide, methotrexate, and 5-fluorouracil (CMF) for 12 months after mastectomy.[2] In 1976, Bonadonna et al published their two-year results confirming a statistically significant benefit in relapse-free survival for patients with positive axillary lymph nodes treated with twelve cycles of these drugs (CMF).[3]

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Accordingly, in 1976 our private practice oncology group in Phoenix began treating appropriate referral patients with CMF after informed consent was obtained. Recently, the office charts of 40 women who initiated CMF adjuvant chemotherapy between 1976 and January 1979 were examined. One patient underwent excisional biopsy with axillary node dissection while all others underwent modified radical mastectomy or a more extensive procedure. Extent of disease was defined by the International TNM Staging System as primary tumor of T_1 or T_2 extent and positive axillary nodes without known metastases.[4] Confirmation in all patients of the above was through reports of operation, pathology, history, physical exam, standard hematological and biochemical profiles, chest roentgenograms, and bone scans. Nearly all patients also underwent liver-spleen scanning.

All patients completed 12 cycles of CMF by February 1980. The CMF schedule was based on the original report of Bonadonna et al,[3] with dosages and treatment delays determined by the treating physician. Twenty-six patients also received radiation therapy before chemotherapy, including the chest wall and regional lymph node fields in all cases and the internal mammary chain in patients with a medial lesion. Doses were usually 5000 rads/field using Co^{60} sources or 4 MeV or 16 MeV linear accelerators.

Following completion of treatment, patients were seen at regular intervals. Appropriate testing was done for symptoms or signs suggesting relapse. Relapses were confirmed by radiological, radionuclide, or pathological means.

Statistical analysis of benefit was carried out by generating relapse-free survival (RFS) curves from a historical data base stored at the University of Arizona for patients who had undergone surgery alone and comparing these curves to carefully matched patients treated with CMF in Phoenix. Comparisions were made by the log-rank method.[5] Using the full calculated dosages of CMF reported by Bonadonna et al[3] as 100%, the percentage of actual total dose administered for the 12 courses of CMF in Phoenix was calculated for each patient. All patients in the Phoenix series received either low doses (<55%, 30 patients) or intermediate doses (55%-84%, 10 patients) rather than full doses (>85%). Comparison of RFS curves was made for the following characteristics: dose of CMF, tumor size, number of positive axillary lymph nodes (1-3, >3), menopausal status, and the use of postoperative radiotherapy.

Table 1 describes the clinical and pathological features of the 40 Phoenix CMF-treated patients who tended to be post-menopausal and have more than 3 involved axillary lymph nodes.

Comparison of relapse-free survival for carefully matched groups of patients failed to reveal any effect of menopausal status or difference between low-dose CMF (<55% total dose) or intermediate-dose CMF (55%-84%) on the relapse rate. For patients with 1-3 positive axillary nodes, no effect of adjuvant therapy was apparent in relapse-free survival, with or without postoperative radiotherapy, compared to surgery alone. All of these patients received either low-dose CMF (< 55% total dose) (11 patients) or intermediate-dose CMF (55%-84%) (4 patients).

Table 1
Distribution of Patients According to Initial Clinical and Pathological Features

Feature	CMF	XRT + CMF	Total
Age (years)			
<40	3	3	6
40-49	3	8	11
50-59	5	11	16
60-69	3	4	7
Menopausal Status			
Premenopausal	4	5	9
Postmenopausal	9	22	31
Tumor Size			
T_1(≤2.0 cm)	5	10	15
T_2 (2.1-5.0 cm)	9	16	25
Positive Axilliary Nodes			
1-3	9	6	15
>3	5	20	25

Figure 1 presents relapse-free survival for patients with more than 3 positive axillary nodes. Relapse-free survival for patients who received CMF alone was not significantly different from that for patients who underwent surgery alone. However, the group of patients who received postoperative radiotherapy and CMF experienced significantly fewer relapses than comparable patients undergoing only surgery (p=0.005). Among the 20 patients in this group, 14 received low-dose CMF (<55%) and six received intermediate-dose CMF (55%-84%).

The original Milan trial was a benchmark in demonstrating that adjuvant combination chemotherapy can reduce the recurrence rate and improve survival in patients with node positive breast cancer.[3] The initial flush of enthusiasm was tempered by apparently diminished effectiveness in postmenopausal women as longer follow-up of the original study occurred.[6] However, recent reanalysis of the Milan data has shown an important dose-response effect of adjuvant CMF.[2,7,8] Patients who received nearly full-dose CMF (>85% of calculated doses) showed marked improvement in relapse-free survival at 5 years, regardless of menopausal status.[7,8] Patients who received <65% of full doses experienced relapses similar to those who underwent surgery alone.[8] Patients receiving doses of 65%-84% experienced intermediate benefit. This dose-response effect was seen in our

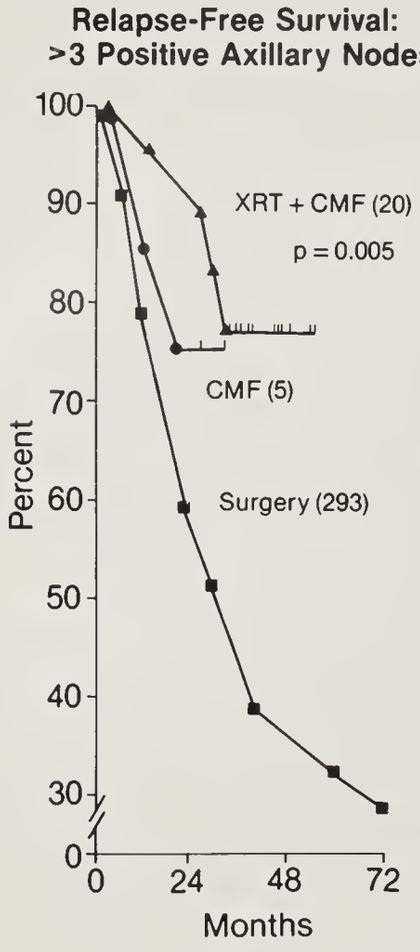


Figure 1

Relapse-free survival curves for patients with more than 3 positive axillary lymph nodes. Marks indicate individual patients at risk at particular times. The difference between patients receiving CMF + radiotherapy and controls (surgery alone) is significant (p=0.005). The difference between patients receiving CMF alone and controls is not significant.

patient population as well. All patients received <85% of full doses of CMF and three quarters of the patients received less than 55% of full doses. Accordingly, the relapse-free survival of patients with 1-3 positive nodes or >3 positive nodes without postoperative radiotherapy (Figure 1) was not significantly different from that for patients undergoing surgery alone.

However, our population of 20 patients with more than three positive nodes who received postoperative radiotherapy followed by low-or intermediate-dose CMF experienced freedom from relapse which was significantly better than that for patients undergoing surgery alone (p=0.005) (Figure 1). These results are different from those of Cooper et al[9] who observed that the relapse-free survival of 27 patients with >3 positive nodes who received radiotherapy followed by the 5-drug Cooper regimen (CMF plus vincristine and prednisone for 1 year) was significantly worse than the excellent relapse-free survival observed in 73 patients who received chemotherapy without radiotherapy.

This analysis of results of adjuvant chemotherapy in a private oncology practice in Phoenix reaffirms the need for practicing oncologists as well as clincial investigators to administer chemotherapy at full or nearly full doses in order to see therapeutic benefit. This study also suggests that a role for postoperative radiotherapy in the adjuvant setting should be further studied. A prospective study of adjuvant chemotherapy with or without radiotherapy is underway in Tucson.[10]

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Radiology

Case of the Month No. 47

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Editors:
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History

A 59-year-old male with bronchogenic carcinoma was admitted with nausea and weight loss. His last course of chemotherapy had been completed one month earlier. The upper GI was normal but endoscopy revealed mild gastritis. Despite the use of cimetidine and anti-emetics, the patient's nausea persisted. A computerized tomographic study of the abdomen was performed. Figure 1 is a slice taken above the level of the kidneys. Figure 2 is taken through the upper poles of the kidneys, which are indicated by the arrows. What is your diagnosis?



Figure 1

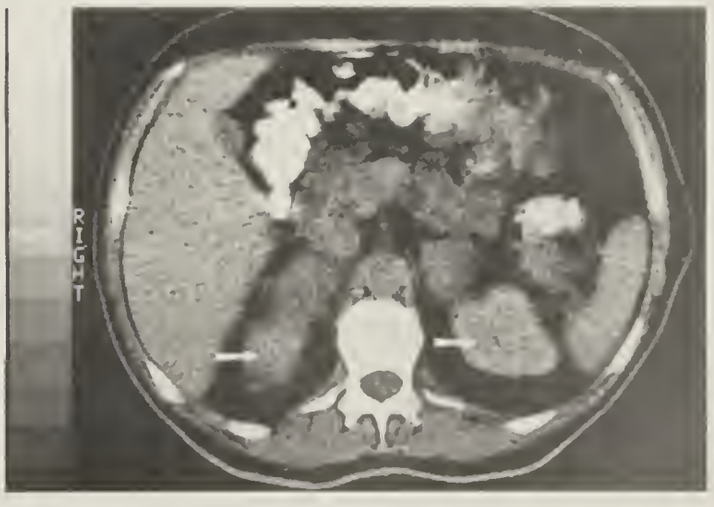


Figure 2

From University of Arizona Health Sciences Center, Department of Radiology, Tucson, AZ 85724

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Observation of the Alert, Conscious Patient With Closed Head Injury

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John Hughes, M.D.

Editor:
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Abstract

In a retrospective study of 96 patients admitted to the University of Arizona Health Sciences Center because of mild cerebral concussion due to closed head injuries between January 1975 and March 1977, only three patients developed neurological complications. Two of these were elderly, but no statistically significant correlation was established between age and occurrence of complications. Radiographic evidence of skull fracture was found in 7 patients, but none developed complications. Since only three percent of the patients studied showed evidence of neurological sequellae after injury, modification in criteria for hospitalization of head injured patients may be appropriate.

The management of closed head injury has varied throughout history, ranging from a form of trephination in the Neolithic period[1] to blood-letting and "watchful neglect" in later eras.

In recent years history, physical examination, skull x-rays, ultrasound examination, cerebral angiography and intracranial pressure monitoring have been used to aid the clinician in estimating the severity of closed head injuries and determining the need for surgical

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Radiology Continued

Answer: Bilateral Adrenal Metastases

The CT scan shows large bilateral adrenal masses. These were confirmed at autopsy to represent metastases from the patient's primary lung tumor.

Metastatic spread to the adrenals occurs in about one-third the cases of bronchogenic carcinoma. Such adrenal involvement is frequently bilateral. Other less frequent sites of metastatic involvement by lung cancer include liver, bone and brain. The adrenals are also favored foci for metastatic dissemination of breast, gastric, colonic and pancreatic neoplasms. The nausea and weight loss seen in this patient resulted from adrenal insufficiency, i.e., Addison's Disease.

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Table 1
Age and Sex Distribution of Patients Admitted with Closed Head Injuries

Age (years)	Male	Female
0-5	17	9
6-10	2	9
11-15	7	3
16-20	9	8
21-25	11	1
26-30	1	4
31-40	4	2
41-50	3	1
51-60	0	1
61-70	1	0
71-80	1	1
80	0	1

Table 2
Duration of Loss of Consciousness Following Injury

Time (Min.)	No. of Patients
0	*26
0-1	34
1-2	23
2-3	9
3-4	2
4-5	1
5-10	1

*15/26 patients with no history of loss of consciousness were 0-5 years of age.

intervention. All of these techniques are of some value. However, one principle that has remained constant in recent years has been hospital admission for observation of those patients with a history of significant loss of consciousness or retrograde amnesia.[2-4] This study of the aftermath of head injuries in a series of patients has been conducted in an effort to establish criteria for such hospital admissions.

Method

The clinical records of all patients admitted to the University of Arizona Health Sciences Center with a diagnosis of closed head injury, between January 1975 and March 1977, were reviewed. There were 260 such patients; those with severe systemic injuries, basal or depressed skull fractures, cerebrospinal fluid rhinorrhea or obvious severe cerebral injuries, e.g., coma or lateralizing neurological signs were excluded from the study. After these exclusions, there remained 96 patients diagnosed as mild cerebral concussion with a history of either retrograde amnesia, loss of consciousness, or both, who were alert and oriented on admission, able to respond fully to verbal commands and without focal neurological deficit. We attempted to correlate factors of age, sex, mechanism of injury, degree of loss of consciousness, presence of skull fracture and duration of observation required with incidence of subsequent complications in these patients.

Results

Mechanism of injury was classified in three categories: 1) motor vehicle accidents (53%); 2) falls (40%) and 3) direct blows to the head (7%). Average age was from 21-25 years in males and 16-20 years in females (Table 1). Radiographic studies of the skull performed on all 96 patients at the time of admission demonstrated a 6.8% incidence of fracture, including one patient with a fracture across the middle meningeal artery. The majority of patients were unconscious for less than two minutes with 30% experiencing no loss of consciousness at all (Table 2). All patients were alert and oriented to person, place and time, fully responsive to verbal commands and had no focal neurological abnormalities following a 2-4 hour observation in the Emergency Room. Children who were included in this study were alert, responsive to stimuli and appeared "normally active" to their parents.

Ninety-eight percent of the patients studied were discharged within three days of admission without any evidence of neurological deterioration. However, three patients did develop significant complications of their head injuries. None of these patients had a skull fracture. Two elderly patients deteriorated later on the day of admission. These two patients, one 76 years of age and the other 82 years of age, became disoriented 8-12 hours after admission although no focal neurological findings were observed. An ultrasound head scan was obtained which revealed midline shift consistent with subdural hematoma. Both patients returned to their pre-injury neurological status following operative decompression by exploratory burr holes. A 23-year old man was readmitted five days after discharge with complaints of increasing headaches and stiff neck. No intracranial hematoma was found although blood in the cerebrospinal fluid obtained by lumbar puncture indicated that a traumatic subarachnoid hemorrhage had occurred. There was no evidence of bacterial meningitis.

The duration of loss of consciousness, duration of retrograde amnesia and presence of skull fracture could not be correlated with the duration of hospital stay or occurrence of neurological deterioration in any of the patients. The fact that the patients who developed subdural hematomas were elderly was not statistically significant but was consistent with increased risk in this age group. Patients who remained in the hospital for more than 72 hours did so either because of disposition problems or persistent headaches rather than neurological complications.

Discussion

The neuropathology of cerebral concussion has not been studied in detail since it is not ordinarily a fatal condition. Concussion had been defined as traumatic unconsciousness due to physiologic impairment of brain

function. However, one study of head injury patients who died of other causes describes "tearing of axons in the brain stem and subcortical white matter."[5] This damage is permanent and can be cumulative with repeated injuries,[6] although it is usually subclinical and does not require hospital admission. Acceleration-deceleraton injury is generally associated with bilateral, diffuse brain injury occurring at the time of impact. However, loss of consciousness may not occur, and extensive focal brain damage may be present without it.[7]

In those patients who have been unconscious, return to consciousness without focal neurologic deficit has often occurred by the time of arrival at the emergency room. The problem of whether or not to admit these concussed patients has been handled in various ways. The accepted policy in many hospitals is admission for observation of all patients who have lost consciousness from head trauma.[8] This practice does provide a setting in which neurological changes may be rapidly identified. Such observation often seems unnecessary, but, in a few patients, delayed neurologic deterioration can occur, and a means of identifying these patients is required if all cases of concussion are not to be hospitalized.

One criterion employed in identifying those patients with mild head injury not requiring hospitalization has been a finding of intact mental status at the time of first examination.[9] It appears that this basis for screening has been the most reliable method in the past.

In a study of 201 victims of road-traffic accidents Overgaard[11] determined that unconsciousness and post-traumatic hypertension in older patients and abnormal motor activity in young patients were the most important signs of unfavorable prognosis. In another study of 3500 minimally head injured patients Jones[9] found only a 1% incidence of continued symptoms or neurological complications; all cases were initially discharged from a hospital emergency room as not in need of further observation.

However, one should hasten to add that patients harborning an epidural hematoma may also be altert on admission during a "lucid interval."[4] Fortunately, the incidence of epidural hematoma is only 2% of head injuries, and those patients who develop hematoma have a 90% incidence of skull fracture. There is also a higher incidence of fractures across a meningeal artery or venous sinus in this group of patients.[4,10] Such patients with skull fracture, who are at a greater risk of having an epidural hemorrhage, should therefore be considered as mandatory hospital admissions.

Recent studies concerning routine skull films in mild head injury patients have demonstrated that a large proportion of this patient population need not even be x-rayed since very little correlation could be made with the appearance of x-rays and the occurrence of further neurological deterioration.[12,13] A 1971 report by Bell and Loop[14] projected that up to $15 million per year could be saved by deferring or altogether eliminating skull x-rays in patients who are deemed unlikely to have fractures on the basis of their history and initial clinical examination. Skull radiographs were obtained in all of our patients because of history of traumatic unconsciousness, and this study was not designed to evaluate the indication for x-ray examination. However, similar efforts could be directed toward reducing the number of needless admissions for observation of mild closed head trauma cases in view of rising hospital costs.

In this retrospective review of 96 hospitalized cases of mild head injury only three patients developed complications and two of these required elective surgical treatment. No patient deteriorated rapidly under observation or suffered delayed onset of permanent neurological deficit. During the period of this study we found no other cases of patients who had been discharged directly from the emergency room for observation at home who required later admission due to development of complications.

Therefore, the results of our study do seem to substantiate the principle that many minimally head injured patients who are alert after a mild cerebral concussion and show no evidence of focal neurologic deficit or deterioration of mental status, severe headache, emesis or seizures could be managed at home with observation by a reliable person rather than by hospitalizaton. However, infants, the elderly, patients who have been unconscious for more than 3-5 minutes and those whose home situation does not permit careful, reliable observation and rapid return to the hospital should be admitted for a brief period of hospital

Table 3
Head Injury Instructions

After injury to the head, the following events can occur once the patient is at home. If any of these occur, call your doctor or bring the patient back to the hospital: Dr. _____
Phone: _____

1. **Inability to remain awake or unconsciousness that cannot be interrupted by awakening the patient.**
2. **Vomiting.**
3. **Severe or worsening headache.**
4. **Weakness or paralysis of arm or leg or loss of balance when walking.**
5. **Slurred speech or inability to speak or understand.**
6. **Blurry or double vision or blind spots.**
7. **Change in the size of one or both pupils.**
8. **Bleeding or fluid drainage from ear or nose.**
9. **Convulsions.**
10. **Abnormal behavior, mental confusion or memory loss. In children under four years, allow the child to sleep but awaken and check every 1-2 hours for the first 24 hours after the injury.**

The following things are normal and to be expected after head injury:

1. **Mild headache.**
2. **Mild nausea.**
3. **Mild dizziness.**
4. **Mild unsteadiness.**

observation (1-3 days). Patients with skull fractures crossing the middle meningeal artery or a venous sinus should also be admitted for observation as should patients with evidence of hemotympanum or cerebrospinal rhinorrhea due to basal skull fracture. Use of these criteria might considerably reduce the cost of medical care by eliminating an average charge of $300/patient day without increasing the risk to the patient of undiagnosed complications of mild head injury.

We do advise, however, that careful explanation of the nature and signficance of signs and symptoms of intracranial bleeding be provided to these patients and their families or other responsible individuals if home observation is recommended. Distribution of an instruction sheet (Table 3) which lists the danger signs, provides an hourly log or check list and includes the name and telephone number of the responsible physician or emergency facility is advisable in order to verify the instructions given and to insure compliance.

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State of the Art

Current Concepts of Cutaneous Melanoma

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Editor:
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Abstract

Malignant melanoma is an important neoplasm to the dermatologist, oncologist and generalist. By heeding several clinical clues early detection will improve prognosis. The incidence of melanoma is rising and its relationship to sun exposure has become more firmly established. Surgical treatment may be curative in certain patients and either chemotherapy or immunotherapy may be effective in advanced disease. Future research in melanoma will focus on the importance of hormonal receptors and the usefulness of the retinoids in therapy.

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Malignant melanoma, if not detected early can be a devastating disease. Our ability to treat advanced disease remains unsatisfactory. There is evidence that in certain parts of the United States and the world that the incidence of melanoma is increasing. Because of these facts, the most important aspect of treatment of melanoma is in its early detection, when lesions may be curable by surgical excision.

In the last decade, emphasis has been placed on the characterization of the earliest clinical changes in pigmented skin lesions indicating malignancy. It is clear that the clinician must be aware of the earliest clinical features of melanoma if we are to improve survival in this malignancy.

Clinical Features

Malignant melanoma can be divided into four major types: lentigo maligna melanoma, superficial spreading melanoma, nodular melanoma and acral-lentiginous melanoma. These types have characteristic clinical or histopathologic features as well as different biological behaviors (Table 1).

Lentigo maligna melanoma generally arises in a pre-existing lentigo maligna which is a slow growing tan macular lesion on the sun exposed skin surfaces of the elderly, most often the face. Often there are areas of regression giving this lesion an irregular appearance. Development of areas of melanoma in a lentigo maligna are often suspected by the appearance of raised brownish-black papules and plaques in an otherwise

macular lesion. These lesions have a long radial growth phase, growing intraepidermally for years before invasion occurs.

Superficial spreading melanoma is the most common type accounting for 70% of all melanomas. It occurs generally during middle life and can appear on any body area. These have a shorter radial growth phase than lentigo maligna melanoma and so tend to become invasive earlier. The major clinical characteristics of early malignancy include variegation of color, irregularity of border, and irregularity of surface.

Variegation of color is probably the most helpful characteristic. Benign pigmented lesions of the skin tend to have a uniformity of pigmentation. True variegation includes shades of red, white, and blue ("flag sign") mixed with tan, brown or black.[1] The color changes occur in a haphazard arrangement and may include all possible shades or hues from the mixture of these colors. These color changes often occur first in the center of lesions, and are associated with loss of normal skin markings.[2] Bluish hues may be the most reliable indicator of malignancy and should not be ignored. White areas should also arouse suspicion and usually indicate areas of partical regression of the tumor. Incontinence of pigment outside the margins of the tumor is also an important clinical sign.

Often these lesions will have an irregular border, with an indented notch.[1] Change in the size and color of pigmented lesions may occur early. The presence of itching was present in 25% of a large series of early melanomas and is thus a helpful diagnostic clue.[5] Early melanomas may be associated with surrounding halos of hypopigmentation. In melanoma, the halo is irregular in shape and the melanoma lies eccentrically with the halo. In benign halo nevi, the pigmented lesion lies in the center of the hypopigmentation.[2] The appearance of multiple halo nevi in later life should provoke a search for a melanoma.[2]

Nodular melanomas have a very short radial growth phase and invade early, usually having significant deep invasion by the time of detection. Irregularity and nodularity are constant features of this type of melanoma. Ulceration and bleeding are late signs. Ulceration is an adverse prognostic sign, with markedly decreased five year survival regardless of tumor thickness.[6]

Acral-lentiginous melanoma occurs in blacks as well as other dark-skinned races and appears on the palms, soles, nail beds and mucous membranes. Clinically these may resemble lentigo maligna melanoma but differ in that it becomes invasive early in its course and does not appear on chronically sun-exposed areas. These tumors may show areas of nodularity as they advance, as well as white areas of regression.[7] Occurrences under the nail plate begin as irregular areas of hyperpigmentation, often occurring in longitudinal streaks, and must be differentiated from benign melanocytic nevi.

Familial melanoma is a newly described syndrome. These lesions have been shown to develop from preexisting melanocytic nevi.[8] Clark et al described 37 patients with familial melanocytic nevi that averaged 1 cm in size and had haphazard brown, black, pink and white pigmentation. There were also morphologic difference from nevus to nevus in the same patient. Histologically these are compound nevi with atypical melanocytic hyperplasia and a lymphocytic infiltrate. They are clearly premalignant in these patients. This has been called the B-K mole syndrome, named after the first family described. Immunologic abnormalities including low T and B cell counts in both affected and unaffected family members have recently been described.[9] The relationship between these abnormalities and melanoma pathogenesis remains to be clarified.

Multiple primary melanomas are frequent enough to demand a complete skin physical examination in any patient with malignant melanoma. Recent data suggest that approximately 5% of all patients with melanoma will have a second primary lesion, 45% of which are diagnosed within the first year and 32% of which occurred synchronously.[10]

Table 1
Clinical Features of Melanoma

Type	Clinical Characteristics
1. Lentigo maligna melanoma	Occur on sun-exposed areas of elderly patients Slow radial growth Irregular tan plaque with white macules and brown papules
2. Superficial spreading melanoma	Favors sun-exposed sites Variation of color-white, blue, red hues, and pigment incontinence Irregular border with notching Irregular surface
3. Nodular melanoma	Rapid growth Irregular pigmented nodule Ulceration and bleeding of tumor
4. Acral lentiginous melanoma	Occurrence on hands and feet in dark-skinned races Irregular pigmented macules which develop nodules Irregular pigmented bands of nails when the nail is involved

Histopathology

Perhaps the greatest advances in the last decade in the management of melanoma are the combined histopathological staging criteria set forth by Clark[11] and Breslow.[12,13] It is clear that both depth of invasion and tumor thickness are related to prognosis.

Clark initially reported level of invasion as an important prognostic factor.[11] Prognosis decreased as depth of invasion increased. He divided the levels of invasion into 5 levels. Level I is an *in situ* melanoma with all tumor cells above the basement membrane in the

epidermis. Level II represents invasion of malignant cells into the upper papillary dermis. Level III is invasion to the junction of the papillary and lower (reticular) dermis. Level IV is invasion into the reticular dermis and Level V is invasion into the subcutaneous fat (Table 2).

Breslow used somewhat different criteria to stage melanoma. He found that tumor thickness as measured by an ocular micrometer correlates better than depth of invasion with prognosis.[12,13] He showed that tumor thickness was directly proportional to the cross sectional area of the tumor which was related to the incidence of metastases and survival (Table 3). Lesions less than 0.76 mm thickness regardless of level of invasion all have an excellent prognosis with almost no incidence of recurrence or metastases after simple excision.

Epidemiology

Recent data indicates that the incidence and mortality rates of melanoma are increasing by 3-9% per year,[14] especially in young people. The increases have been seen at selected body sites, particularly on the lower legs in females and the trunk in males. It has been suggested that sun exposure is a direct etiologic agent. There is a significant body of evidence to support this hypothesis. Recent reports from Israel,[15] South Wales[16] and the Nordic countries[17] all confirm the relationship between sun exposure and melanomas. It has also been shown that peak incidence increases are related to peaks of sunspot activity.[18] It may be that current habits of recreation and clothing have contributed to the increasing incidence of melanoma.

Meteorologic factors and geography are also important. Schreiber et al[19] in a study conducted in Arizona demonstrated an increasing incidence from 6.5 to 27 per 100,000 over a 10 year period. This increase was most pronounced in the 50-69 year old age group, with most lesions occurring on sun exposed areas. The incidence in Arizona was 7 times the average expected in the United States. The factors proposed to be important in Arizona include genetic factors, meteorologic factors (clear days, less daytime cloudiness, low humidity, high average temperatures) as well as geographic factors (high altitude, low latitude and decreased atmospheric ozone).

Racial differences in the incidence of melanoma also support ultraviolet exposure as a contributing etiologic factor. In a study of 59 population-based cancer registries, Crombie[20] demonstrated that non-white populations have a significantly lower incidence of melanoma than whites. Africans had a higher incidence than Asians but this was only because of the high incidence of acral lentiginous melanoma, thought not to be related to ultraviolet exposure. Increased skin pigmentation seems to be a protective factor in the development of melanoma on exposed body sites.

Treatment of Melanoma

Surgical Therapy. The advance in histopathological staging using thickness and depth of invasion as criteria has helped to standardize the surgical approach to malignant melanoma.

Table 2
Clark's Histological Staging of Melanoma

Level 1	Tumor confined to epidermis (in situ melanoma)
Level 2	Tumor invasion into upper papillary dermis
Level 3	Tumor invasion to junction of papillary dermis and reticular dermis
Level 4	Tumor invasion into the reticular dermis
Level 5	Tumor invasion into the subcutis

Table 3
Breslow Histologic Staging of Melanoma

Tumor Thickness	Prognosis
<0.76 mm	90% 5 year disease-free interval
0.76-1.5 mm	70% 5 year disease-free interval
>1.50 mm	64% with regional lymph node dissection
>1.50 mm	31% without lymph node dissection

It is generally accepted that excision with simple closure is adequate therapy for all lesions less than 0.76 mm thick. The width of the margin and the need for grafting should be determined by the anatomic location of the tumor.[21,22] The confusion in the literature over appropriate surgical treatment comes when both Clark's levels and thickness are used simultaneously or interchangeably.

Several authors have suggested not only wide excision but also elective lymph node dissection be done for Clark's level III-V, lesions measuring 0.9 mm or greater and all nodular melanomas.[23]

Others have recommended excision and regional lymph node dissection for all Clark's levels III-V and all melanomas greater than 1.5 mm thick.[24] It has also been suggested that prognosis in melanoma is determined by the number of lymph nodes histologically involved, node palpability and site of the melanoma. In patients undergoing lymphadenectomy, it was found that thickness of the tumor was less important prognostically than these other factors.[25]

The evidence for the efficacy of elective lymph node dissection in improving survival is equally confusing. Some have reported that higher cure rates were obtained in both Clark's level III and IV melanoma when elective lymphadenectomy was done with wide excision.[26] In the only prospective randomized large scale study comparing excision alone with elective regional lymph node dissection of clinically uninvolved nodes no statistical difference in survival of the two groups was demonstrated.[27]

To synthesize all of this disparate data into a workable clinical formula is difficult. It seems that eliminating the Clark's level of invasion and using only tumor thickness as a criterion would eliminate some confusion. All tumors less than 0.76 mm should have a simple excision, the margin of which should be at least 2 cm if possible.[22]

No lymph node dissection is necessary in these lesions. Those lesions 0.76-1.50 mm should have a 3-5 cm margin of excision with grafting as necessary. Lesions greater than 1.5 mm in thickness should also have wide local excision. Regional node dissections for these last two groups is debatable, advocated by some and not by others. Certainly, clinically involved nodes should be resected en bloc with the involved tumor.[22]

Immunotherapy. Immunotherapy with BCG has been shown to be of some value in the treatment of melanoma. The cutaneous lesions of malignant melanoma may undergo regression following BCG injection. In order for BCG to be effective the patient must have normal skin test reactivity to standard test antigens.[28] BCG seems to exert its action by a nonspecific stimulation of the immune system.

The effectiveness of BCG in treating regional lymph node metastases has also been established. It seems to have an inhibiting effect on microscopic metastases, and also seems to work better in patients with small tumor loads.[29] The effectiveness of BCG in visceral metastases has been shown to be less helpful, and may aggravate symptoms.[30] The complications of BCG immunotherapy include uveitis, vitiligo,[31] fever, influenza-like symptoms, liver dysfunction[32] and even death.[33]

Long term follow-up of melanoma patients treated with BCG suggests that prolonged treatment with BCG scarification may exhaust the immune system and may ultimately increase the risk of dissemination of the malignancy. Helander et al[34] have shown that with prolonged use of BCG (100-140 weeks) skin test reactivity to PPD and response of lymphocytes to mitogens tend to decrease, often prior to clincial dissemination of disease. These immunologic parameters should be followed in all patients receiving BCG and may be useful in predicting dissemination.

Attempts at using specific active immunotherapy have recently been reported. Seigler et al[35] used sequential immunization with irradiated neuraminidase treated melanoma cells along with BCG. The results are promising but are too preliminary to make firm statistical statements regarding efficacy.

Transfer factor has been used in widely disseminated melanoma with limited success. Gonzalez et al[36] successfully used transfer factor in patients with melanoma metastatic to lung. They suggest that transfer factor may be helpful in those patients with a small tumor load and a tumor responsive to immunologic manipulation. Further studies will be necessary to fully delineate the role of immunotherapy in malignant melanoma and its use presently should be considered as experimental.

Chemotherapy. It is beyond the scope of this paper to review all of the various chemotherapeutic protocols that have been used in the treatment of melanoma.

Several studies have examined the response of melanoma to chemotherapy alone or chemoimmunotherapy. Chemoimmunotherapy with dimethyl triazeno imidazole carboxamide (DTIC) plus BCG showed a remission rate in lymph node metastases of 55% compared with 18% in those treated with DTIC alone. The duration of remission and survival were significantly longer for patients treated with chemoimmunotherapy than for those treated with chemotherapy alone.[37]

Others have suggested that adjuvant (treatment in the absence of overt disease) chemotherapy using multiple drugs in addition to surgery may improve survival.[38] Still others have shown that adjuvant immunotherapy with BCG is more effective than adjuvant chemotherapy with DTIC.[39] All of these therapeutic modes deserve further careful study in the treatment of malignant melanoma.

Future Directions. Recently, attention has turned to the possibility that endocrine factors have an influence on malignant melanoma. Survival in females has for many years been known to be better than males. Several reports have noted the exacerbation of melanoma during pregnancy. It appears then that a minority of

patients with melanoma may have a hormonally responsive tumor.[40]

Significant numbers of patients with melanoma have estrogen binding activity in the tumor. Some have evidence of progesterone binding, androgen binding, and glucocorticoid binding. The conclusions drawn by Neifeld and Lippman were that there is a small subset of patients whose melanomas are responsive to steroid hormones and that steroid binding presence or absence does not correlate with response to hormone therapy.[40]

The relationship between estrogen receptors in benign nevi and malignant melanoma may give us further insight into this hormone receptor concept. In a recent study, estrogen receptors were found in 41% of benign nevi from melanoma patients.[41] Thirty-seven percent of the melanomas from this group also had estrogen receptor activity. No nevi from normal persons had estrogen binding activity. The suggestion is made that estrogen receptor activity may be an acquired molecular alteration associated with the transformation of a benign nevus into a melanoma. This may represent a biological marker for potential melanoma patients.

The retinoids are a group of compounds chemically related to vitamin A with diverse pharmacologic effects. Animal and in vitro studies have suggested that certain of these compounds have antineoplastic effects. The exact mechanism(s) of action are unclear but several different theories have been proposed including an immunostimulatory effect, an effect on the differentiation of anaplastic cells and possibly a direct cytotoxic effect on the neoplastic cells.

We have recently treated two patients with numerous cutaneous metastatic melanomas with topical vitamin A acid and achieved a complete remission in one patient and a partial response in the other person.[42] It is possible that this class of compounds given systemically as well as topically may be of value in the treatment of melanoma. Studies are currently in progress to further assess their efficacy.

References available upon request.

In Memorium



Clarence G. Salsbury, M.D. 1886-1980

Dr. Salsbury's life work included that of a missonary, surgeon, hospital builder and administrator, founder of the first nurses' training program for American Indians, peerless administrator of the Arizona State Health Department, physician, and withal a friend of man. Now he has joined the saints!

To describe this many-faceted physician would require a concerted effort by all organizations and medical activities he so valorously served. Salsbury was—if nothing else—an activist, in the true sense of the term.

Born in Canada of New England Loyalist (Tory) ancestory, he decided early on, at high school, to be a medical missionary. On completion of his high school work at the Union Missonary Institute in Brooklyn, New York, he entered the College of Physicians in Boston. After he acquired an M.D., he interned at the New York Lying-In Hospital, married Cora Burrows, a fellow student at the Missonary Institute, and accepted an assignment for missonary duties in Hainan, China.

Next, came twelve years as a medical missionary and builder of hospitals and a high school. He literally built a high school using a diesel-powered saw mill and redwood timber he had imported from the States.

The Communist takeover forced him and his fellow missonaries to leave China. Then it was back to the States and New

York, where he served temporarily in an administrative position with a central missonary board. This was not his cup of tea, so he finally agreed to serve temporarily at a mission hospital and school at Ganado on the Navajo reservation. This was to be temporary; he still wanted to be assigned overseas. It turned out to be quite "temporary." He spent the next twenty-five years of his life there with his wife, Cora, rebuilding, renewing, and establishing many innovative programs at this hospital and school. When he came, the hospital was a 15-bed ramshackled adobe building without patients. When he left they had a modern brick hospital, the first nurses' school established for the training of Indian girls, and a first-rate secondary school and high school. The hospital was the first to be accredited in Northern Arizona by the American College of Surgeons. Some may not be aware that the accreditation program was inaugurated by the American College of Surgeons, and that a Phoenix physician, E. Payne Palmer, Sr., was said to have introduced the resolution before a meeting of the College to inaugurate this program.

Noteworthy is the fact that some Eskimo girls were also accepted to the nurses' school, and during World War II, Japanese girls were accepted for nurses' training when they were not so accepted in any other place in the United States.

The following statistics are from his records of 1948: "We produced 40 tons of alfalfa and 23 tons of oats. Three and 3/4 tons of vegetables were raised for campus consumption. The total of 285 chickens gave us 4,275 eggs, and we ate 420 pounds of chicken which had stopped laying.

"Our dairy cows produced 7,373 quarts of milk. We also consumed 8,051 pounds of pork which we raised ourselves and 2,370 pounds of beef.

"The number of patients admitted during the years was 2,252 and they spent 3,938 days in the hospital. We had 3,927 dispensry patients. The operations performed were 415, births 185."

Salsbury had definite ideas about the inadequate Indian Bureau and perhaps one of the greatest chances for the improvement of that government bureaucracy was plowed under when Dewey failed to make it as President. Salsbury was slated to be director of Indian Affairs.

On May 25, 1950, he and his wife retired from their work at Ganado, and he remarked, "Our temporary assignment has come to 23 years. With our years in China before that, we had a 36-year career as missonaries. Yet it didn't seem long. It was difficult to try to convince myself that I was 64 years old."

Almost immediately he was offered a position with the State of Arizona Health Department as chief of the Bureau of Preventive Medical Services which had just been created. As he related it, nobody seemed to know exactly what his duties were. But he soon outlined them since he felt that he should do something to earn his income which was $7,400 a year, the biggest income of his life to that point. He found that only about half of the physicians in the State were filing communicable disease reports, so he offended them by asking them to do their paper work properly. He offended some other people by talking publicly about syphillis and gonorrhea, and others were startled when he took out after open privies and venereal disease control in general. He learned by accident that a Mexican national had died of typhoid in a work camp in Casa Grande, so he took out after the primitive methods and unsanitary conditions amongst the agricultural work force and offended the ranchers. A code was drawn up by the health department for labor camps, published and proclaimed the law of the State. The health department sent out enforcers. There was a great difficulty around Marana where the ranchers claimed they couldn't afford that sort of accommodations for their workers, but as Salsbury noted, they quickly raised money when three of them were brought to court. They suddenly had an enormous amount of funds available for their defense.

He was appointed health commissioner to succeed Dr. Ward. Dr Salsbury continued to jump from the frying pan into the fire with his campaign for better living conditions for farm workers, for better disease reporting, for better case findings of venereal disease and tuberculosis. All of this bore fruit but gained him the enmity of the ranchers and many other segments of the population, including the State Legislature.

By now he was 75 and he had another retirement with many awards and accolades, but one he apparently appreciated as much as, if not more than, any other was by Robert Price, then president of the Maricopa County Medical Society. Dr. Price said, "He was outspoken when he saw suffering humanity and was ready to publicize in every way possible the unhealthy conditions he found. His path was not a rosy one. He found opposition to many of his public health programs—at times from the legislature, on occasion from influential lay groups, and even from members of the medical profession in a few instances."

During this time the County Medical Society had established a Clarence G. Salsbury Medal in his honor to be awarded for outstanding service. Salsbury himself was awarded the Thomas Dooley Medal for valor of service at the close of his public health career.

"The ones who live are the ones who struggle, the ones whose soul and heart are filled with high purpose. Yes, these are the living ones." This description by Victor Hugo certainly personifies the life and work of Clarence G. Salsbury.

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Public Relations —

Taking Medicine's Story to the People

Public relations, according to one definition, is the "art or science of developing reciprocal understanding and goodwill between a person, firm or institution, and the public".

ArMA needs a strong, consistent public relations program. It needs an experienced PR staff member to direct that program.

- We have to build and maintain strong contacts with press, radio and TV staff personnel. We must establish ArMA—and therefore the physicians of Arizona—as a prime resource for accurate information on health care issues and medical news. Our news information office should be a place where reporters can call to verify a story or get the background they need for an article; a source that is credible, easy to contact, fast to reply.
- We must bring doctors, society officers and staff together informally with representatives of local media so they can frankly discuss socioeconomic aspects of medical practice and health care, as well as pertinent community problems.
- We should be prepared to state ArMA's position on medical issues clearly and forcefully. We must try to anticipate problems and be able to react swiftly when news breaks affecting Arizona physicians.
- We ought to publicize doctors' involvement in community service activities and look for additional ways in which volunteer physicians can meet specific health needs.
- We can emphasize the physician's involvement in health education and his concern for the public by producing radio and TV programs featuring local doctors speaking on medical topics. We should continue to reach newspaper readers with health columns and expand that service by offering the columns to employee publications of the major industries in the state. We ought to be producing explanatory health and cost brochures and posters for doctors' offices.
- We have to coordinate ArMA's public relations activities with those of the county societies. We should stand ready to serve as a PR consultant to the county societies or to individual physicians in those counties interested in developing active programs.
- We need to create a quick, effective communication system which will get information about ArMA activities to our membership.
- We need to make public relations an integral part of all our planning. A staff PR person experienced in writing and graphics, creative, sensitive to the moods of the public and our members, could be a tremendous asset in planning all aspects of association activity and in preparing the printed materials we use in those projects.

It will, of course, take more than one person to make a public relations program effective. We'll need doctors willing to serve as spokesmen for medicine, ready to answer questions forthrightly. "In the past," says David Zeitlin, director of communications for the Los Angeles County Medical Association, "the inaccessibility of physicians bred a kind of distrust. If reporters know they can get straight no-nonsense, authoritative, credible information and opinions quickly, their attitude will be civil and even friendly."

We'll need doctors willing to give time to community projects. "When physicians help solve community problems, their credibility rises tremendously," according to James R. Hickox, executive director of the Harris County (Texas) Medical Society, in an article written for *Medical Executive* magazine.

We'll need doctors who will take the time to help us build coalitions—find allies—in the business community, institutions and other organizations also treatened by government regulation. A good PR program requires we work not only with the media, but that we tell medicine's story directly to the people of our community.

Most important, we'll need doctors who remember that the most effective public relations is to continue to meet our patients' need in those daily encounters at the office and in the hospital. Stu Spencer, a nationally-known political and public relations consultant, said it best. "The sum total of a patient's experiences—how long he's waited, the attitude of the staff, the caring of the physician—will to a major degree determine the profession's image".

John T. Clymer, M.D.
President, ArMA

The Pulmonary Clinical Nurse Specialist

The pulmonary clinical nurse specialist makes significant contributions to the clinical, research and teaching programs of the Division of Respiratory Sciences in the College of Medicine at the University of Arizona. The major purpose of this article is to describe the clinical roles of these nurses in patient care. Within this description, however, it is necessary to clarify the background and education of nurses with the title, pulmonary clinical nurse specialist (PCNS). Medicine has long had a generally accepted system for identifying and defining the various roles under the broad title, "physician;" namely, specialty and sub-specialty designation by board certification. Until recently the title "nurse" was not differentiated much beyond administrative titles (e.g., head nurse and staff nurse). Today, however, there are many new roles within nursing—the nurse specialist, nurse practitioner, and clinical nurse specialist—all of which function differently. Because many of these roles are still evolving, their descriptions and definitions have not always been clearly defined and communicated. The result has been confusion about what a title means and the expectations of the person and the role he or she fulfills.

The pulmonary clinical nurse specialist has a master's degree in nursing with advanced knowledge, skill and competence in the care of persons with actual or potential pulmonary dysfunction. The PCNS may be based in a variety of settings (e.g. acute care hospital, clinic, group practice, etc.). Regardless of setting, the major emphasis of the PCNS is clinical practice. Included in this clinical practice are direct patient care, consultation to physicians and others regarding pulmonary care measures and consultation, and educational programs for other nursing personnel. Providing continuity of care and coordinating the various aspects of patient care are also important functions of the PCNS.

It must be stressed that the PCNS represents an expanded role within nursing. It is not a role which competes with the physician; rather, the two roles are complementary. The physician's emphasis is medical diagnosis and formulation of a general plan of care. The PCNS's emphasis is the implementation of a plan of care which considers the unique needs of the individual patient, family, and significant others. Some patients may need to see the physician more frequently than the nurse, others will see the nurse more frequently, and some will need to see both professionals routinely. There is overlap of the two roles, but the emphasis of each is different. To give an example, the physician sees a patient with chronic bronchitis and emphysema. The patient has been placed on a program of bronchodilators. The nurse then assesses the patient's life style, disease, and treatment. Some of the questions to be answered include: is the medication schedule effective and appropriate; are actions and side effects of medications understood; how does shortness of breathing affect the patient's life style—what modification in activity or breathing pattern would be helpful; is cough effective or should different cough techniques be taught; are other airway care techniques needed; are patient/family coping mechanisms effective; does the patient have sufficient knowledge to assist in long-term self-surveillance? As the PCNS identifies problems, means of intervention must be delineated. Some may involve changes in the pharmacologic intervention and are done in collaboration with the physician (e.g., different type of bronchodilator, change in dosage, etc.) Many of the interventions emphasize patient/family education and counseling. Some of these interventions are instituted via individual patient-nurse interaction, some may involve group sessions, and some may involve referral to other sources (e.g., physician, social worker, etc.)

The PCNS provides "caring" and is able to help the patient deal with the day to day problems precipitated by disease and treatment. The expertise of the PCNS also allows confidence that complications and/or new medical problems will be identified and referred to the physician. Thus, the PCNS provides the patient with an additional access route to the health care system.

In summary, the goal of the PCNS is the same as that of the physician - to help the patient live as fulfilling a life as possible within the limitations of disease. The attainment of that goal is facilitated by the nurse-physician collaboration in providing care. For the staff and patients of the Respiratory Division, this goal contributes to better overall individualized care.

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Correspondence

On September 18, 1980, the *Phoenix Gazette* published an article, the headline of which read: "Health Ploys Costing Millions, official says." The article implied widespread fee splitting on the part of Arizona physicians. Letters of objection were submitted to the papers and an explanation requested from the Department of Health. That explanation follows:

Mr. Bruce E. Robinson
Executive Director
Arizona Medical Association

Dear Mr. Robinson—

This letter is in response to your request for clarification of an article entitled "Health Ploys...," which appeared in the Phoenix Gazette on Thursday, September 18, 1980.

I am sure you are aware that public employees in our Department obtain information via confidential consumer complaints concerning questionable practices taking place in the health care industry. As a result of this exposure, staff may formulate personal points of view or opinions which are not necessarily those of the Director or management of the Department. When questioned by the media, staff may offer personal opinions in response to questions.

It has never been the policy of the Department to prevent employees from speaking to the media. We hope that the employee will indicate that the point of view presented is his and not necessarily the official position of the Department. We also hope that the reporter will present the story accurately. In the article in question, the view expressed by Mr. Webb primarily related to certain questionable practices uncovered in other states under their Medicaid programs and possible similar practices occuring in Arizona.

Since the Medicaid program has not been implemented in Arizona, the Department's responsibility relating to alleged abuses is found in A.R.S. § 36-407.D, which states, "The licensee (of a health care institution) shall not personally or through an agent offer or imply an offer of rebate or fee-splitting to any person regulated by provisions of Title 32 or Title 36, Chapter 17." Enforcement of this statute rests with the Director of the Department of Health Services. Regulation of alleged physician to physician practices is under the jurisdiction of the Board of Medical Examiners. See A.R.S. § 32-1401.10. p. and t.

As for the Department's official activity in the area of alleged abuses, during the past several months we have had internal discussion regarding the definition of the statutory terms 'fee-splitting and rebates.' We believe these terms should be defined in regulations which should be developed with the involvement of all interested parties before our enforcement action in this area is initiated.

We hope this will clear up any misunderstanding that may have occurred.

Suzanne Dandoy, M.D., M.P.H.
Director, Arizona Department
of Health Services

ArMA members should be reminded that in Medical Memos #108 3/17/78 legal counsel cautioned the membership about possible problems with percentage based contracts. A Department of Health Services committee is to meet to discuss this matter. You will be represented at that meeting and we will keep you posted.

Editor—

Seminars in Dermatology, by Dr. M. M. Schreiber ("Acute Sunlight Damage to the Skin," *Arizona Medicine* Vol. 37, No.4 pp 254-256), contains at least three errors of fact. Dr. Scheiber states:

1. "Water vapor - Particles of water act as mirrors giving more reflected rays." Not true. Water *vapor* is an invisible gas, consisting of dispersed free molecules of H_2O, which actually *absorbs* some wave lengths of light. In no sense can it "mirror" light. *Condensed* water in the atmosphere or frozen water crystals (ice), as in cirrus clouds, can reflect as well as refract light rays, but these are certainly not water vapor.
2. "Time of Year - In the summer the sun is closer to earth so the U V radiation is stronger." An egregious error. In the summer the earth is actually two to three million miles *distant* from our sun than in winter. (December distance, 91.5 million miles *vs* June, 94.5 million miles). The fact is that the *tilt* of the earth's axis is such that a higher concentration of photons is absorbed per unit area in summer than in winter.
3. "Latitude - The closer the equator, the stronger the U V rays. Basal cell carcinoma and squamous cell carcinoma incidence in the United States doubles for each *3'48"* decrease in latitude (265 miles)." *(Emphasis added)* The designation 3'48" means 3 minutes, 48 seconds, or about 3.47 statute miles. Surely what is meant is 3°48', which is 3 degrees, 48 minutes of latitude, or about 262 miles. Even so clarified this is an example of an unknowable statistic being carried out to an absurdly precise point. Even if one ignores racial population blend, cover, and a host of other variables, one can only crudely extrapolate the geographic incidence of skin cancer. For example: The Anglo-Nordic ranchers of northern Nevada doubtless have more such tumors than do South Louisiana "Cajun" fishermen, despite the closer geographic proximity of the latter group to the equator.

Robert W. Blair, M.D.
Mesa, Arizona

Editor—

In answer to Dr. Blair's comments regarding my article "Acute Sunlight Damage to the Skin:"

Item 1 - In quoting just one of the many references, *Dermatology in General Medicine*, Second Edition (The most recent standard text on Dermatology) page 945. See reference No. 5 of the article.

Shorter wavelengths of UV light are scattered by atmospheric molecules and particles much more than longer wavelengths. Much of the UV radiation reaching the earth's surface is that part scattered downward by the atmosphere. This sky radiation can account for 30-50% or the total solar U-V radiation reaching the earth on a clear day, and that is why one can receive a sunburn even if one is exposed in a shadow.

Infrared radiation is more strongly absorbed by clouds (consisting of *water droplets*). In certain circumstances (on a cloudy day), the total U-V irradiance on the ground may exceed that occurring on a cloudless day. Such an event may be produced by strong downward *reflection* from the clouds, distributed in such a manner that the sun is unobstructed.

Perhaps it might have been clearer to Dr. Blair to say *water droplets*, of which clouds are composed, rather than *water vapor*.

Item 2 - Dr. Blair is certainly correct in his statement that the sun is closer to the earth in winter than the summer.

Item 3 - Yes, the 3'48" should have been 3°48'. This was a typographical error.

I'm not quite sure why Dr. Blair is refuting this statement, but I can give him two very reliable references supporting this by acknowledged experts in the field of photobiology. 1) Auerbach H: Geographic variation in incidence of skin cancer in the United States. *Public Health Rep.* 76:345-348, 1961; and 2) Cooper AGS: The contribution of radiotherapy to the problem of skin cancer in Queensland. *Acta Radiol* 188 (Suppl.): 61, 1969.

There are many recognized factors both human and environmental contributing to skin cancers, which I would be happy to go over with Dr. Blair

if he would contact me. It is universally acknowledged by epidemiologists, oncologists, and dermatologists who study skin cancers, that there is indeed (supported by hundreds of statistically epidemiologic studies) a higher incidence of all types of skin cancer - basal cell and squamous cell carcinomas, and malignant melanomas in populations living closer to the equator, as compared to those living further from the equator.

Dr. Blair's example of northern Nevada ranchers having more skin cancers than Louisiana "Cajun" fisherman is not a valid one. I know of no statistical studies supporting this; but assuming he is right, and he probably is—because the ranchers of northern Nevada, are more than likely, light-skinned Caucasians, who spend much more time out in the sun, at a higher altitude, and with significantly more clear days, compared to the Louisiana Cajuns. Having lived 12 years in the Louisiana Cajun country, I know that these people have darker skin and do not have as much sun exposure as ranchers in the West, even though they are closer to the equator. This in no way invalidates the known and supported fact that skin cancer incidence is higher in people living closer to the equator.

Michael M. Schreiber, M.D.
Tucson, Arizona

ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, August 16, 1980, convened at 1:18 p.m., Suresh C. Anand, M.D., Chairman, presiding.

Election of Vice-Chairman Chairman 1982 Annual Meeting

Stuart I. Holtzman, M.D., was unanimously elected as Vice-Chairman of the committee, with the understanding that he will serve as Chairman of the 1982 Annual Meeting.

Review of 1980 Meeting

Evaluation Forms

Summary of the 1980 Annual Meeting Scientific Program Evaluation Form. *Received.*

During the review of the summary provided to the committee, it was determined that the evaluation forms should be made more available throughout the course of the meeting and moderators be requested to urge the participants to complete and return the forms to enable the committee to make a more complete evaluation.

Resurvey of CME Program

Report of the Resurvey of the Continuing Medical Education Program of the Arizona Medical Association. *Received.*

News Coverage

Recap of News Media Coverage. *Received*

1980 Annual Meeting Attendance Report

The 1980 Annual Meeting Attendance Report, together with a recap of members registered by specialty and the following report of attendance at previous years' meetings were reviewed by the committee.

Year	Attend	Members	%	Location
1980	441	2705	16.3	The Pointe Resort - Phoenix
1979	603	2688	22.4	Safari Resort - Scottsdale
1978	421	2554	16.5	Marriott - Tucson
1977	498	2556	19.4	Hyatt Regency - Phoenix
1976	704	2471	28.5	Safari Resort - Scottsdale
1975	459	2393	19.2	Braniff Hotel - Tucson
1974	660	2232	29.6	Safari Hotel - Scottsdale
1973	654	2079	31.5	Safari Hotel - Scottsdale
1972	550	1866	29.5	Safari Hotel - Scottsdale
1971	390	1745	22.4	Safari Hotel - Scottsdale
1970	377	1669	22.6	Safari Hotel - Scottsdale

Comments and Suggestions

Letters of Suresh C. Anand, M.D. (5/16/80), John C. Clymer, M.D. (6/16/80), Clyde W. Kurtz, M.D. (5/5/80) and the ArMA Auxiliary. *Received.*

Attendance at Scientific Sessions

Report regarding number of attendees at each of the scientific sessions during the meeting. *Received.*

1981 Plans

Moving of Dates

Board of Directors' letter date 1/11/80 approving moving of dates to "off-season". *Received.*

May 20-24, 1981

Letter from The Pointe Resort agreeing to the new dates of May 20-24, 1981, for the annual meeting, with favorable offseason room rates. *Received.*

In discussing this item, the committee determined that all promotional material for the 1981 Annual Meeting should greatly stress the lower room rates which would be available.

ArMA Auxiliary Plans

Joyce Newman, President of the ArMA Auxiliary, outlined tentative plans for the physician/spouse portion of the annual meeting, which would be held on Friday, May 22, 1981, the theme for which would be "Facets of Communication" and would consist of a general session on all phases of communications, which would then break into three, or possibly four, workshops with more specific topics.

It was moved and carried to approve the physician/spouse program to be sponsored by the Arizona Medical Association Auxiliary on Friday, May 22, 1981, "Facets of Communication," which would consist of a general session on all phases of communications, breaking into workshops for discussions on "Communication and Sexual Attitudes in Marriage," "Parent-Teenage Communications," and "Persuasive (and Defensive) Communication." The concept of a possible fourth workshop was also approved, to be left to the discretion of the Auxiliary.

It was moved and carried to approve the Auxiliary's selection of guest speakers for the physician/spouse program, which selection would be greatly dependent upon speaker availability, with the exception that ArMA staff would seek the participation of Mortimer T. Enright, Director of the AMA Speakers and Leadership Programs section, and additionally request that Mr. Enright present a "Speaking Effectively" seminar on Thursday afternoon, May 21, 1981.

Mrs. Newman also presented to the committee three possible logos for use in the program and promotional materials for the annual meeting and, after a review, the committee determined that the choice of logo be left to Mrs. Newman.

Moderators

Dr. Chisholm addressed the committee relative to more personal contact between moderators and guest speakers and it was the consensus of the committee that the initial instruction to the moderators include a sample letter with the suggestion that each moderator write a similar letter to each of those

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Each capsule contains
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antianxiety/antisecretory/antispasmodic

for adjunctive therapy of duodenal ulcer* and irritable bowel syndrome*

Librax®

Please consult complete prescribing information, a summary of which follows:

Indications: Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis
Final classification of the less-than-effective indications requires further investigation.

Contraindications: Glaucoma, prostatic hypertrophy, benign bladder neck obstruction, hypersensitivity to chlordiazepoxide HCl and/or clidinium Bromide.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**

As with all anticholinergics, inhibition of lactation may occur.

Precautions: In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

Adverse Reactions: No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated, avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered, isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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Motrin® vs codeine...

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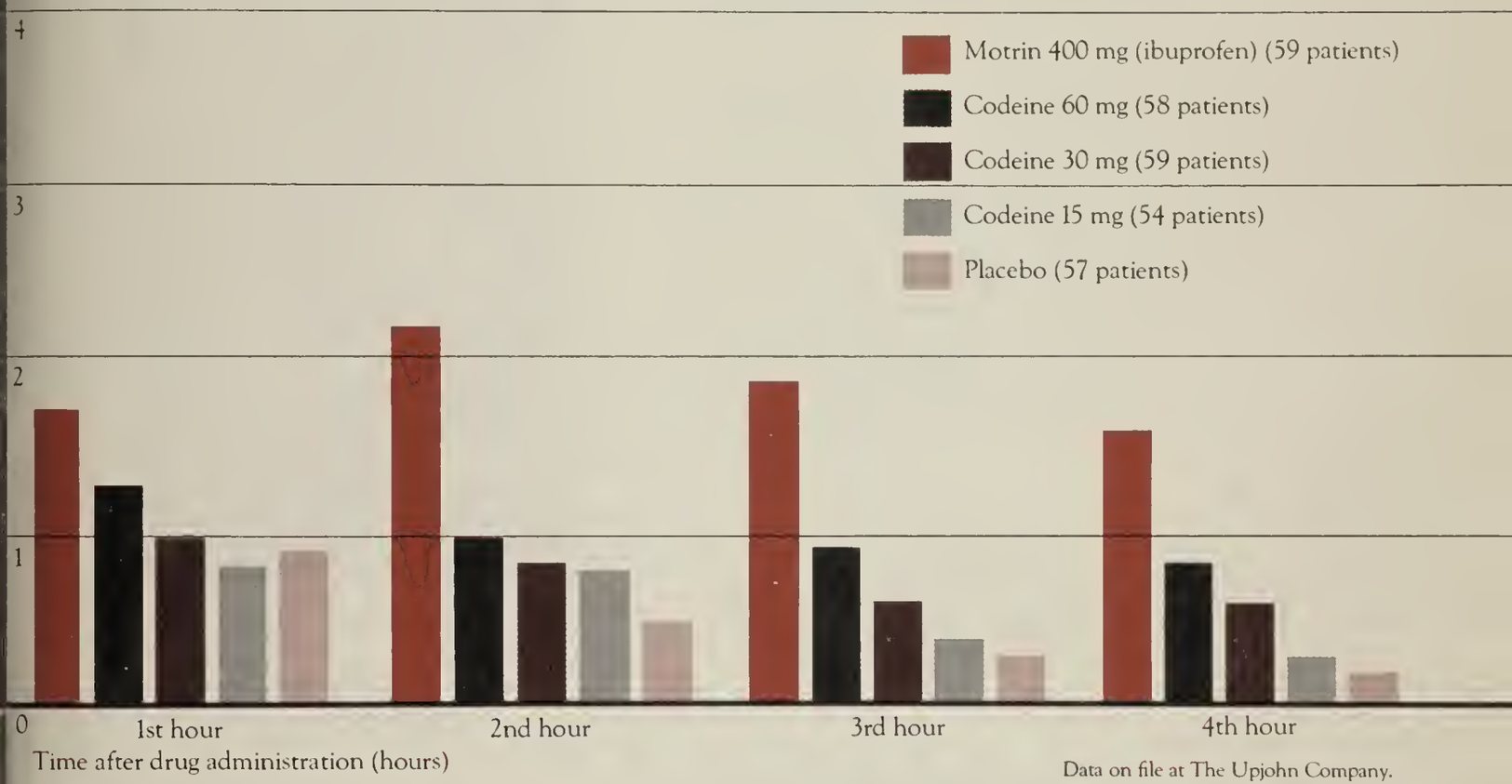
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compare the analgesic effect

Motrin (ibuprofen) 400 mg tablets provided greater relief of pain than codeine in a double-blind, randomized clinical study of 287 patients. *Motrin* was significantly more effective ($p < 0.01$) than codeine 60 mg at the 2-, 3- and 4-hour intervals...significantly more effective ($p < 0.01$) than codeine 30 mg, codeine 15 mg, and placebo at all intervals.

Degree of pain relief—mean scores

4 = Excellent relief 3 = Good relief 2 = Fair relief 1 = Poor relief 0 = No relief



Data on file at The Upjohn Company.

One tablet q4-6h prn pain

A well-tolerated, nonnarcotic prescription for mild to moderate pain

Motrin® 400 mg TABLETS

ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming • Acts peripherally
- Relieves pain rapidly • Indicated in acute and chronic pain • Well tolerated
- The most common side effect with *Motrin* is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen)
now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Relief of mild to moderate pain.

Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation.

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid arthritis and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.
Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

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May 1980

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Thanks to you, it works. For all of us.

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guest speakers to whom he was assigned. Additionally, the committee determined that the host system would again be instituted and that early communications to the speakers would include brochures and informational materials about the Phoenix area and the activities available to facilitate preplanning of the speakers and their families.

ArMPAC Banquet

Mr. Robinson informed the committee that the Arizona Medical Political Action Committee had suggested that it not hold a banquet in conjunction with the 1981 Annual Meeting and, following discussion,

It was moved and carried, at the suggestion of ArMPAC, to cancel, for 1981, the ArMPAC Banquet normally held in conjunction with the Annual Meeting.

Steak Fry

The committee discussed the social function usually held in the form of a western steak fry and it was determined that a change should be made with respect to the theme of this function.

It was moved and carried that the theme for the social function be changed to something other than a steak fry, possibly a luau, and that such determination, together with finalization of plans, be left to the discretion of Bruce E. Robinson.

It was moved and carried that, due to the cancellation of the ArMPAC Banquet, the social function above-referred to, be held on Friday evening, May 22, 1981, with the President's Banquet on Saturday evening, May 23, 1981, leaving the Thursday evening, May 21, 1981, a free evening.

Scientific Program -
Number of Concurrent Sessions

The committee discussed at great length the continuation of running four concurrent scientific sessions and made the following determination.

It was moved and carried that, as in the past, four rooms would be held for use during scientific sessions of the 1981 Annual Meeting, with concurrent sessions being run in three rooms only and the fourth room held for special use in filling additional programs, use by specialty societies, or possibly the holding of CPR courses.

Selection of Speakers - Recommendations of Specialty Societies

The committee considered the recommendations of various specialty societies concerning guest speakers to be requested to participate in the scientific portion of the annual meeting and, relative thereto, determined that:

1. Michael D. Ellis, M.S., Associate Director of the Poison Control Center, University of Texas Medical Branch at Galveston, be requested to provide sessions on the following suggested topics: Diagnosis and Treatment of Poisoning; Poisonous Plants; Common Drug Poisons. Should he be unable to participate, a like request would be made of Barry H. Rumack, M.D., Director of the Rocky Mountain Poison Center in Denver.
2. Victor Fuchs, Ph.D., at Stanford University, be requested to participate by providing a session on Medical Care Rationing, with the possibility of building a panel to discuss similar problems within Arizona, which would be scheduled to immediately follow and be a continuation of Dr. Fuchs' program.
3. To request a presentation on Sports Medicine by Robert L. Larson, M.D., President of the American Orthopaedic Society for Sports Medicine, from Eugene, Oregon. It was determined to schedule a full day's program around Sports Medicine oriented subjects, drawing from the guest speakers who, in fact, agreed to participate, as well as local physicians well-versed in this field.
4. Rene Cailliet, M.D., of Pacific Palisades, California, be requested to participate, possibly as primary speaker in the day-long Sports Medicine portion of the program, the feeling being that, with his knowledge in the field of rehabilitative medicine and his being well-versed in many subjects, he would be able to provide presentations on various topics. Should Dr. Cailliet be unavailable, it was agreed that Richard S. Materson, M.D., of Houston, Texas, would then be contacted; and, additionally, Dennis H. Harris, M.D., would be requested to be a local speaker.
5. It was determined to request Charles Kerr, M.D., a Tucson psychiatrist, in addition to his participation in the Auxiliary program, to speak on the psychological apsects of sports injuries.
6. The committee was apprised that Richard Friedenberg, M.D., of the University of California at Irvine was already planning to be in Phoenix on Thursday and Friday, May 21 and 22, 1981, to speak to various groups, including the Arizona Chapter, American College of Radiology, and it was agreed that he be contacted to see if his schedule would allow his participation during scientific sessions.
7. It was determined that the Arizona Chapter of the American College of Surgeons be allocated an entire morning for scientific presentations, with ArMA to finance one guest speaker and the College to finance the balance of speakers, all of whom would be selected by James J. Berens, M.D. Dr. George Burdick agreed to chair an ad hoc committee to contact Dr. Berens regarding the action of the committee and report back to the committee regarding final selection of speakers and topics at the earliest opportunity.
8. James W. Mosley, M.D., of the Rancho Los Amigos Hospital in Downey, California, who has been involved on a consulting basis with the CDC-Hepatitis Laboratories in Phoenix, to make a presentation regarding hepatitis, with a possibility of contacting local physicians knowledgeable in the disease to add additional sessions to the program.
9. It was determined to request local physicians, Phillip J. Rubin, M.D., Harry W. Hale, Jr., M.D., and J. Burr Ross, M.D., to participate as guest speakers, seeking their suggestions as to topics.

Generally, it was agreed to request each speaker to provide one to one-and-one-half hours of presentations, either in total time or half-hour slots.

Dr. Kurtz advised the committee that he would discuss with the Arizona Urological Society the possibility of developing a program and report back to the committee with the particulars as soon as possible. Likewise, Dr. Holtzman indicated that it was entirely possible that the Arizona Society of Physical Medicine and Medical Rehabilitation would again want to sponsor a portion of the scientific program as it had during the 1980 Annual Meeting.

Offers of Other Speakers

It was moved and carried to not accept the offer of the Fitness Motivation Institute of America dated July 30, 1980, to provide a guest speaker.

It was moved and carried to accept the offer of Central Diagnostic Laboratory to provide Joseph K. Portaro, Dr.Ph., to speak on the topic of HLA Typing and Disease Susceptibility.

Solicitation of Pharmaceutical Companies

The committee determined that guest speakers provided by pharmaceutical companies in the past had contributed greatly to the quality of the program and

It was moved and carried that a general solicitation of pharmaceutical companies be made for speaker sponsorship, with the final selection of speakers and their topics to be left to the discretion of the chairman.

Breakfast Panels

It was moved and carried that breakfast panels during the 1981 Annual Meeting would be reduced to one, to be held on Saturday, May 23, 1980, the topic for which would be determined by the chairman at a later date and, additionally, that only coffee and rolls be served at such function, to enable attendance at no charge.

Disaster Preparedness

The committee determined that the subject of disaster preparedness was one of great interest and, following

discussion,

It was moved and carried that James M. Hurley, M.D., attempt to arrange a presentation, hopefully to be coordinated into the auxiliary schedule, on disaster preparedness and report back to the committee with all particulars at the earliest possible date for finalization of plans in this regard.

1982 Meeting

The committee received for its information the letter of the Camelback Inn dated 4/29/80, a report on negotiations with the Arizona Biltmore, and a letter from The Pointe Resort dated 6/4/80, confirming that it could, in fact, host the 1982 annual meeting May 26-30, together with the Board of Directors' action of 8/9/80 confirming those dates and that location, subject to the action of the committee.

It was moved and carried approving the dates of May 26-30, 1982, for the holding of the 1982 Annual Meeting at the Pointe Resort in Phoenix, with other alternatives to be evaluated for the 1983 meeting, with the recommendations to come before the committee for determination during its next scheduled meeting.

Other Business

Residents/Interns

The committee discussed at great length the desire to bring about a greater involvement in the annual meeting by the residents and interns within the state and it was determined that, via letters to the various resident/intern programs advising of the location, date changes, etc., these physicians be encouraged to participate.

Registration Fees

The committee again discussed the problem of member registration fees and the feeling that the elimination of such fees, perhaps by a general dues increase, might be a solution to promoting greater attendance at annual meetings.

It was moved and carried to request the Finance Committee to review ArMA's present dues structure and consider, for recommendation to the Board of Directors, a dues increase which would include a sufficient amount to allow elimination of ArMA member annual meeting registration fees.

Breakfast Sessions

It was determined, because of the time availability, to advise the state specialty societies that they might wish to consider holding breakfast sessions during the 1981 Annual Meeting.

Awards

The committee determined that it would give further consideration to possibly changing the categories for awards to promote a more balanced competition among the participating physicians.

EXECUTIVE COMMITTEE

A meeting of the Executive Committee of the Arizona Medical Association, Inc., held at 810 West Bethany Home Road, Phoenix, Arizona, on Friday, September 19, 1980, a quorum being present, convened at 6:58 p.m., John T. Clymer, M.D., President and Chairman, presided.

Steering Committe
Research and Education Committee

Dr. Earl Baker conducted a brief meeting of the Steering Committee of the Research and Education Committee, whose mandate was to research and educate the members of the House of Delegates on facts relating to de-unified membership with the AMA, which would again be considered in meeting on November 1, 1980, in Resolution No. S-1-80. Dr. Baker and his committee discussed at great length the compilation of data from other states as well as opinions of various medical groups within Arizona, after which basic guidelines were established for the meeting of the entire committee to be held on October 11, 1980, as well as determinations made as to distribution of the final informative materials.

News Article
Phoenix Gazette

The committee inquired of Dr. Dandoy as to the basis for the news article which appeared in the September 18th edition of the Phoenix Gazette. She advised the committee that the Department was checking all possible records to determine if there were any facts to substantiate the statements contained in the article; and, should there be none, some type of letter of regret would be requested from the Department to the Association.

Arizona Department of
Health Services

Arizona Crippled Children's Hospital

Dr. Dandoy updated the Executive Committee regarding the progress on the Crippled Children's Hospital, advising that the Futures Committee had, in fact, completed its activity and would hold no more meetings and that the Pediatric Society was continuing its endeavors in regard to continuation of the services by this facility.

Eye Drop Bill

The committee was advised that the panel for the "eye drop bill" was moving ahead with the hearings, the first of which had recently been held in Flagstaff, and others now being scheduled throughout the state.

Emergency Medical Services

In conjunction with earlier attempts, Dr. Dandoy advised that there would be legislation introduced during the upcoming session which would bring the state's emergency medical services under the Department of Health Services and she requested that the Association make every effort to assist in the passage of the bill.

Request for Funds to Attend
AMA Meeting

The committee reviewed a request from Ronald P. Spark, M.D., Chairman of the Media Committee of the Pima County Medical Society, dated August 28, 1980, for financial assistance to attend a Health Reporting/Radio-TV Workshop being sponsored by the AMA October 2-4, 1980.

It was moved and carried that, while the Association strongly supports individual participation in AMA-sponsored programs, due to budget restrictions, financial assistance for attendance by Ronald P. Spark, M.D., at the Health Reporting/Radio-TV Workshop being sponsored by the AMA October 2-4, 1980, must be limited to an offer by the Association to pay the registration fee of $75.00.

Boddicker Vs Arizona State
Dental Association

Findings of Fact, Conclusions of Law and Judgment entered 8/29/80 in favor of defendants by Federal Court in Boddicker vs. Arizona State Dental Association. *Received.*

October 4, 1980 Meeting Schedule

Schedule of activities for October 4, 1980 in Tucson. *Received.*

Arizona Perinatal Trust

Mr. Robinson reported to the committee that the Arizona Perinatal Trust had now become a legal entity, with trust documents having been executed and funds transferred, and that by reason of the trust document, ArMA agreed to name one member of the APT Board of Trustees.

It was moved and carried to recommend to the Board of Directors that the Association name William C. Scott, M.D., as a member of the Board of Trustees for the Arizona Perinatal Trust.

Other Business

Search Committee

Article published in the 9/13/80 edition of the Arizona Republic indicating members of the search committee to find a replacement for Dr. Suzanne Dandoy as Director of the Department of Health Services, which membership includes Daniel T. Cloud, M.D., William J. Moore, M.D. and Marilyn Heins, M.D. *Received.*

1981 ArMA Committees

Dr. Kurtz requested and received permission to proceed to contact various specialty and county societies with a view to possibly re-structuring ArMA committees for the year 1981.

Video Cassettes

It was determined to investigate the sources and possibilities available for consideration of medical education for

member physicians via video cassettes.

Insurance Discounts

It was determined that legal counsel would be requested to furnish an opinion as to what, if anything, might be done to secure some type of discount through the Mutual Insurance Company of Arizona for ArMA members.

PROFESSIONAL COMMITTEE

Meeting of the Professional Committee of the Arizona Medicial Association held September 17, 1980 at 810 West Bethany Home Road, Phoenix convened at 7:14 p.m., Jospeh W. Hanss, Jr., M.D., Chairman, presiding.

New Members

The Chairman welcomed the new committee members, Dr. DeArmond, Dr. Powell, and Dr. Reuben.

Section on Aging and General Medicine

Definition of Death- AMA Model Legislation

The Resolution "Statutory Definition of Death - American Medical Association Model Legislation" which was tabled by the House of Delegates during the 1980 annual meeting was presented by Dr. Hagan. It was noted that Mr. Jacobson, ArMA General Counsel, had not had an opportunity to review the resolution prior to introduction and expressed concern with its content. After considerable discussion the committee generally agreed with the American Medical Association Model Definition of Death and suggested that it be introduced in the Arizona legislature during the next session.

It was moved and carried to request legal opinion on a statutory defintion of death for Arizona modeled from the American Medical Association and request that the legal opinion and the model legislation provided by the American Medical Association be presented to the Board of Directors for consideration.

Determination of Death in Nursing Homes

Dr. Hagan briefed the committee on the problems that have been encountered when a person expires in a nursing home during the middle of the night and the physician is called out to "pronounce death" as well as sign the death certificate. Since the last committee meeting, there have been amendments to the current Health Department regulations deleting the requirement that a person who dies in a nursing care institution be "pronounced dead" by a physician. It was suggested that the Association sponsor a meeting of nursing home administrators, medical directors, and the Department of Health Services to discuss the problems encountered and seek a resolution.

It was moved and carried that the subject concerning definition of death in nursing homes be referred back to the section on Aging and General Medicine for meeting with all appropriate parties to resolve the problem expressed by the Department of Health Services.

Section of Allied Medical Groups

Optometric Practice Act

Mr. Barnett briefed the committee concerning the "eye drop legislation" adopted by the Arizona legislature in behalf of Optometrist. It was noted that public hearings are being held currently and a report will be submitted to the legislature in January concerning the public acceptability to this legislation. *Received for Information.*

Chiropractic Practice Act

Mr. Barnett briefed the committee on the Chiropractic Practice Act which was adopted during the last legislative session. It was noted that concerns have been expressed with "procedures preparatory and complementary to the adjustment" and legal counsel is looking into the statement as to its meaning. *Received for Information.*

Section on Emergency Care & Disaster Medicine

Responsibilities & Interrelationship of Paramedics and Physicians

Correspondence by Dr. Krentz of February 19, 1980, Dr. VanNostrand of March 6, 1980, and Tony Mitten of March 7, 1980 concerning the responsibility and interrelationship of paramedics and physicians on the scene of an emergency medical event was considered. Dr. VanNostrand was unavailable to comment.

It was moved and carried to table discussion on the responsibility of interrelationship of physicians and paramedics on the scene of an emergency medical event until the next meeting of the Professional Committee.

Section on Health Education

An Early Start to Good Health

The Board of Directors, in meeting of March 8, 1980, reviewed the Professional Committee's recommendation regarding "An Early Start to Good Health" and adopted its recommendation. *Received for Information.*

Section on Poison Control

S.B. 1067 Establishing an Arizona Poison Control System

It was announced that Senate Bill 1067, establishing an Arizona Poison Control System was adopted in accordance with the concerns of the Professional Committee.

Section on Public Health

Communicable Disease Regulations - Rubella Immunity

The Committee received correspondence from the Arizona Hospital Association regarding communicable disease regulations with respect to rubella immunity. It is suggested that there be continuing discussion with the Arizona Hospital Association on this matter in an attempt to resolve the problem without official regulation by the Department of Health Services. The committee endorses this concept and is willing to meet with the Hospital Association.

Section on Safety

Child Passenger Restraints in Automobiles

The legislation introduced the past two sessions, as well as, the American Medical Association Resolution 47 adopted in 1978 supporting legislative activity and minutes of the Governors Highway Safety Council concerning Child Passenger Restraints was presented to the committee of consideration. It was noted that the Association actively supported Mandatory Child Passenger Restraints in Arizona the past two years. Concerns were expressed regarding the implimentation of this type of legislation and the results of Tennessee and Rhode Island. There was also concern expressed regarding penalties toward relatives of children, i.e., grandparents transporting without the proper restraint system.

It was moved and carried to table discussion of mandatory child passenger restraints in automobiles until the next meeting.

Section on Sports Medicine

Resignation of William C. Brainard, M.D.

Dr. Brainard's resignation was accepted with regret. Dr. Hanss reported that Dr. Brainard's suggested replacement, Thomas Foerster, M.D. was accepted by the president and will be presented to the Board of Directors for confirmation on October 4, 1980. *Received for Information.*

Correspondence

Navajo Health Authority

Correspondence was received by the Navajo Health Authority, Office of Native Healing Sciences, concerned with the utilization of traditional medicine and other native medicine in hospitals, nursing homes, etc. on the Navajo reservation. It appears that Navajo patients request traditional Navajo medicine in these facilities and concerns are expressed about the state and/or federal violations. It was determined that the section on Medicine and Religion should review this matter with the proper authorities and report

back to the committee during its next meeting.

Other Business

Pre-marital Examination

Mr. Barnett presented a complaint regarding the cost of pre-marital examinations which would include a complete physical examination. It was noted that County Health Department personnel appear to agree that physicians charge exorbitantly for this service. Staff was asked to discuss the problem with the proper authorities at the County Health Department and determine whether an article should be published concerning this problem in the Medical Memos.

FINANCE COMMITTEE

The meeting of the Finance Committee of the Arizona Medical Association held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, September 27, 1980, convened at 10:01 a.m., John T. Clymer, M.D., President, presiding as Chairman.

1981 Committee Budgets

The committee received and reviewed oral and written requests from the Chairmen of ArMA committees relative to the amounts deemed necessary for each of such committees to function during the 1981 calendar year. Following a lengthy discussion and, in light of the action of the House of Delegates establishing a total 1981 Committee Department Budget in the sum of $69,000.00, the committee took the following action:

It was moved and carried to recommend to the Board of Directors that the 1981 committee budgets be as follows:

500A	Articles of Incorporation and Bylaws	$ 500.00
500B	Benevolent & Loan	6,000.00
500D	Board of Directors	4,200.00
500E	Executive	3,000.00
500F	Finance	500.00
500H	Grievance	-0-
500I	History & Obituaries	-0-
500J	Maternal & Child Health	1,000.00
500K	Health Manpower	800.00
500L	Legislative	18,000.00
500M	Medical Education	3,000.00
500N	Medical Economics	1,000.00
500O	Occupational Health	-0-
500Q	Professional	3,500.00
500R	Public Relations	20,000.00
500S	Governmental Services	2,000.00
500U	Housestaff	1,000.00
500W	ArMA Auxiliary	1,000.00
500X	Physician Rehabilitation	3,500.00
Total 1981 Committee Department Budget		$ 69,000.00

Annual Meeting Registration Fees

The committee reviewed the request of the Scientific Assembly Committee made during meeting on August 16, 1980, that this committee review ArMA's present dues structure and consider a dues increase which would include a sufficient amount to allow elimination of ArMA member annual meeting registration fees, and took the following action:

It was moved carried to recommend to the Board of Directors that the recommendation of the Scientific Assembly Committee to review ArMA's present dues structure and consider a dues increase which would include a sufficient amount to allow elimination of ArMA member annual meeting registration fees be rejected.

Arizona Perinatal Trust

Accounting Services

The committee reviewed the Board of Directors' direction to reconsider provision of accounting services to the Arizona Perinatal Trust after October 4, 1980 cut-off date and, additionally, was advised that the Trust has now been legally established and has funds with which to carry on its work. The committee determined to take no action on the matter.

Board of Trustees

Letter from Ted F. Warner dated Septemer 2, 1980, regarding membership of the APT Board of Trustees. *Received.*

Interest and Scholarships

Board of Directors' action of 8/9/80 regarding interest income used for scholarships. *Received.*

AMA Official Family Briefing A Great Success



Dr. William Payne, Dr. John Kerr, and Dr. Larry Linkner at ArMA's reception for Dr. Lowell Steen, chairman of AMA's Board of Trustees.



Dr. Lowell Steen, chairman of the AMA Board of Trustees, with Dr. Dan Cloud of Phoenix, AMA president-elect.



Joyce Newman, president ArMA Auxiliary and John T. Clymer, M.D., president, ArMA.



Dr. and Mrs. Robert Stark, Dr. Herbert Munhall, Dr. Clyde Kurtz and Mrs. Munhall at the Steen reception.

The medical leadership of Arizona met in Tucson on October 4 to participate in an AMA Official Family Briefing onducted by Lowell H. Steen, M.D., chairman of the Board of Trustees of the American Medical Association and aniel T. Cloud, M.D., president-elect of the AMA.

Drs. Steen and Cloud updated the Arizona leadership on many national and international issues facing medicine, as ell as participating in an active question and answer session. It was a tremendously successful and informative day.

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

NOVEMBER

The Last Act of Living: Dying and Death
November 5, 12, 19. Consecutive Wednesday, Phoeinx Public Library, 12 E. McDowell Rd., Phoenix, AZ. Sponsors: Good Samaritan Hospital, Department of Medicine, Arizona Humanities Council, ASU College of Nursing and Hospice of the Valley. Contact. Ms. Kathy Wyse, Program Coordinator, College of Nursing, ASU, Nursing 362, Tempe, AZ 85281.Approved for 2½ hours of category 1 credit for first two segments.

Rocky Mountain Clinical Geneticists
November 14, 1980. Tempe, Arizona. Sponsor: Genetics Center of Southwest Biomedical Research Institute. Contct: Joan Holman, The Genetics Center, 123 East University Drive, Tempe, AZ 85281.

American College of Physicians Regional Meeting
November 14-15, 1980. Arizona Biltmore Hotel, Phoenix, AZ. Sponsors: American College of Physicians. Contact: Alan Gordon, M.D., 2200 N. 3rd St, Phoenix, AZ 85004. Approved for 9 hours of category 1 credit.

Arizona Pediatric Society
November 14-16. Westward Look Resort, Tucson. Sponsor: U of A College of Medicine & Arizona Pediatric Society. Contact: Faith Carlisi-Smith, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 11 hours of category 1 credit.

"Risk Managment For Safety Professionals"
November 17. Student Union-U of A, Tucson. Sponsor: U of A College of Medicine. Contact: Herschella Horton, Arizona Center for Occupational Safety and Health, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

Alzheimer's Disease & Family Involvement
November 17. Arizona Health Sciences Center, Tucson, AZ Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 5 hours of category 1 credit.

Personal Instruction Course In Cataract Surgery
November 19-22. The Eye Foundation, Phoenix, AZ. Sponsor: The Eye Foundation. Contact: The Eye Foundation, 421 N. 18th Street, Suite 105, Phoenix, AZ 85006. Approved for 30 hours of category 1 credit.

Geriatrics Conference
November 20-21. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Christy Snow, Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours category 1 credit.

The Legal Hurt of Chronic Pain
November 20, 21, 1980. The Mountain Shadows Resort, Phoenix, Arizona. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator. St. Luke's Hosptial, 525 N. 18th Street, Phoenix, AZ (602) 258-7373, Ext. 311. Approved for 14 hours of category 1 credit.

Comprehensive Review in Toxicology
November 20-22. Sheraton-Pueblo Inn, Tucson. Sponsor: Denver Dept. of Health & Hospitals, A.C.E.P., & A.A.F.P. Contact: CRIT, c/o Peter Bryson, M.D, Emergency Department, Denver General Hospital, 8th Ave. & Cherokee St. Denver, CO 80204. Approved for 15 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
November 21-22, 1980. Arizona Medical Assn., Phoenix, AZ. Sponsor: ACEP, Arizona Affiliate-American Heart Assn., & ArMA. Contact: Carol Turner, ArMA, 810 West Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Thrombolytic Therapy
November 25, 1980. Desert Samaritan Hosp., Meas, AZ. Sponsor and Contact: Office of CME, Desert Samaritan Hospital, 1400 S. Dobson, Road, Mesa.

Collective Review - Abdominal Aneurysi Cases
November 25, 1980. Walter O. Boswe Memorial Hospital, Sun City. Sponsor an Contact: Office of CME, Walter O. Boswe Memorial Hospital, 10401 Thunderbir Blvd.Sun City, AZ.

DECEMBER

State of the Art in Critical Care Medicine
December 2, 1980. Desert Samarita Hospital. Sponsor and Contact: Dese Samaritan Hospital, Office of CME, 1400 S Dobson Road, Mesa, AZ.

Controversies in the Management o Coronary Heart Disease
December 3-4. Los Angeles Hilton Hotel, Lo Angeles, Ca.. Sponsor: Lyman A. Brewer I International Surgical Society. Contact: Th Secretary-Treasurer, Lyman A. Brewer I International Surgical Society. Contact: Th Secretary-Treasurer, Lyman A. Brewer, I International Surgical Society, 172 Brooklyn Ave., Box 137, Los Angeles, C 90033. Approved for 14 hours of category credit.

Personal Instruction Course in Cataract Surgery
December 3-6 and 10-13, 1980. The Ey Foundation, Phoenix, AZ. Sponsor an Contact: The Eye Foundation, 421 N. 18t Street, Suite 105, Phoenix, Arizona 8500 Approved for hour per hour category 1 credi

2nd Annual Sports Medicine Symposium
December 4-6. Marriott Hotel, Tucson, AZ Sponsor: U of A College of Medicine Contact: Faith Carlisi-Smith, Office of CME Outreach, U of A Health Sciences Cente Tucson, AZ 85724. Approved for 16 hours o category 1 credit.

International Congress on Ericksonia Approaches to Hypnosis and Psychotherap
December 4-8. Phoenix Civic Plaza Convention Center, Phoenix, AZ. Sponso Maricopa County General Hospital, Phoeni AZ., & Milton H. Erickson Foundation, Inc Contact: Jeffrey K. Zeig, Ph.D., The Milton H Erickson Foundation, Inc., 1935 East Aureliu Ave., Phoenix, AZ 85020. Approved for hou per hour category 1 credit.

Occupational Medicine for the Primary Car Physician
December 6, 1980. Arizona Health Science Center, Tucson, AZ. Sponsor: U of A Colleg of Medicine. Contact: Office of CME Outreach, U of A Helath Sciences Cente Tucson, AZ 85724.

"Endocrine Surgery - Plus" Annual Surgical Conference
December 12-13, 1980. The Pointe Resor Phoenix, Arizona. Sponsor: America College of Surgeons, Arizona Chapte Contact: F. Darwin Zahn, M.D., 333 E. Virgini Ste 201, Pheonix, AZ 85004. Approved for 1 hours of category 1 credit.

LYMAN A. BREWER, III INTERNATIONAL SURGICAL SOCIETY
6TH ANNUAL SCIENTIFIC SYMPOSIUM

on

"CONTROVERSIES IN THE MANAGEMENT OF CORONARY HEART DISEASE"

In Association With White Memorial Medical Center — Loma Linda University School of Medicine

'ednesday, December 3, 1980. 8:00 A.M. to 4:30 P.M. . .and Thursday, December 4, 1980. 8:00 to 4:30 P.M.

The Los Angeles Hilton Hotel

GUEST FACULTY

AVID H. BLANKENHORN, M.D., *Professor of Medicine, University of Southern California School of Medicine.*

YMAN A. BREWER, III, M.D., *Distinguished Physician, U.S. Veterans Administration, Jerry L. Pettis Memorial Veterans Hospital.*

GREG BROWN, M.D., Ph.D., *Associate Professor of Medicine, University of California, Los Angeles, School of Medicine.*

ORTIMER J. BUCKLEY, M.D., *Professor of Surgery, Harvard University Medical School, Boston, Massachusetts.*

REDERICK A. HEUPLER, M.D., *Department of Cardiology, Cleveland Clinic Foundation, Cleveland, Ohio.*

ILLIAM P. LONGMIRE, JR., M.D., *Professor of Surgery, University of California, Los Angeles, School of Medicine.*

RICHARD K. MYLER, M.D., *Associate Clinical Professor of Medicine, University of California, San Francisco, School of Medicine.*

MICHAEL P. STERN, M.D., *Associate Professor of Medicine, University of Texas Health Science Center, San Antonio, Texas.*

QUENTIN R. STILES, M.D., *Associate Professor of Surgery, University of Southern California School of Medicine.*

EDWARD STINSON, M.D., *Thelma & Henry Doelger Professor of Cardiovascular Surgery, Stanford University School of Medicine, Stanford.*

RAELENE L. KINLOUGH-RATHBONE, M.D., Ph.D., *Associate Professor of Pathology, McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada.*

FLOYD D. LOOP, M.D., *Chairman, Department of Thoracic and Cardiovascular Surgery, Cleveland Clinic Foundation, Cleveland, Ohio.*

COURSE DESCRIPTION

ıe 6th annual scientific symposium will present current controversies in the prevention, diagnosis and management of coronary heart sease. A panel of international active clinical investigators will discuss avant garde concepts and clinical findings regarding progress in e prevention and treatment of this disease entity. The course is especially of interest to: the cardiovascular and vascular surgeon, eneral surgeon, cardiologist, angiography internist, family practice physician, cardiovascular and operating room nurse and perfusionist.

CREDIT

HYSICIANS: This program offering is approved for 16 hours Category 1 credit toward the California Medical Association Certificate in ontinuing Medical Education, The American Medical Association Physician Recognition Award, and the American Academy of Family ysicians.

URSES: Approved for 16 contact hours of continuing education. Provider No. 00404, BRN State of California.

MERICAN SOCIETY OF EXTRACORPOREAL TECHNOLOGY:

INFORMATION

EGISTRATION FEES: (Includes luncheons, published symposium proceedings, and refreshment breaks) Physicians $175; Residents, iterns, Nurses, and Others $50. Medical and Nursing Students: Free admission to lectures with letter from Dean.

LEASE MAKE CHECK FOR APPROPRIATE AMOUNT PAYABLE TO: LYMAN A. BREWER III INTERNATIONAL SURGICAL SOCIETY

MAIL TO: Secretary-Treasurer, LYMAN A. BREWER III INTERNATIONAL SURGICAL SOCIETY,
1720 Brooklyn Avenue, Box 137, Los Angeles, California 90033

Workshops on Bones & Joints
9th Annual Seminar on Bone & Joint Disease II
December 12-14, 1980. Marriott's Camelback Inn, Scottsdale, Az. Sponsor: Maricopa County General Hospital. Contact: Alex Newman, M.D., Program Dir., Dept. of Radiology, MCGH, 2601 E. Roosevelt St., Phoeix, AZ 85008. Approved for 14 hours of category 1 credit. Bone and Joint Seminars only, 8 hours.

Surgery
December 13, 1980. Arizona Health Sciences Center, Tucson, AZ Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, U of A Health Sciences Center, Tucson, AZ 85724 Approved for 6 hours of category 1 credit

1981
JANUARY

Eight Annual Symposium
Barrow Neurological Institute of St. Joseph's Hospital, & Medical Center - Controversies in Neurology and Neurosurgery
January 8-10, 1981. La Posada Resort, Scottsdale, Arizona. Sponsor: St. Joseph's Hosptial and Medical Center, and the Barrow Neurological Foundation. Contact: Richard A Thompson, M.D., Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013. Approved for 15 hours category 1 credit.

Postgrad Course in the Diagnosis & Treatment of Headache
January 9-11, 1981. Mariott's Camelback Inn, Scottsdale, AZ. Sponsor: American Assn. for the Study of the Headache. Contact: Am. Assn. for the Study of Headache, 5252 N. Western Ave., Chicago, Ill. 60625. Approved for 15 hours of category 1 credit.

Personal Instruction Course in Catract Surgery
January 14-17 and 28-31. The Foundation, Pheonix, Arizona. Sponsor and Contact: The Eye Foundation, 421 N 18th Street, Suite 105, Phoenix, AZ 85006. Approved for hour per hour category 1 credit.

Ninth Annual Surgical Symposium
January 19-23, 1981. Del Webb's La Posada Resort Hotel, Scottsdale, AZ. Sponsor: Maricopa County General Hospital and Phoenix, Surgical Society. Contact: Phoenix Surgical Society, 333 W. Thomas Rd., Suite 209, Phoenix, AZ 85013. Approved for 31 hours of category 1 credit

Clinical Recognition and Management of Heart Disease 1981
Twenty-Fourth Annual Scientific Session of the American Heart Association, Arizona Affiliate
January 21-24, 1981. University of Arizona College of Medicine, Main Auditorium, Tucson, AZ. Sponsor: University of Arizona College of Medicine, American College of Physicians and the Arizona Heart Association. Contact: Office of CME and Outreach U. of A. Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Common Problems with the Hand & Upper Extremity
January 27, 1981. Walter O. Boswell Memorial Hospital, Sun City AZ. Sponsor and Contact: Dept. of Education, Walter O. Boswell Memorial Hospial, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for hour per hour category 1 credit.

A.A.F.P. Annual Scientific Meeting
January 29-31, 1981. Doubletree Inn Mall, Scottsdale, AZ. Sponsor: Arizona Academy of Family Physicians. Contact: Lee Mahoney, 8534 E. Amelia, Scottsdale, Arizona 85251. Approved for 18 hours of category 1 credit.

FEBRUARY

7th Annual Meeting
Freestanding Ambulatory Surgical Association
February 5-7, 1981. Doubletree Inn, Scottsdale, AZ. Sponsor: Arizona Medical Assn. & the Society of Freestanding Ambulatory Surgical Association. Contact: Brian Dawson, M.D., 1040 E. McDowell Road, Phoenix, AZ 85006. Approved for 16 hours of cateory 1 credit.

Rural Emergency Medicine
"The First 60 Minutes"
February 12 & 13, 1981. Doubletree Inn, Scottsdale, AZ. Sponor: Arizona Chapter American College of Emergency Physicians, AZ Affiliate - Am. Heart Assn. & ArMA. Contact: Carol Turner, ACEP, Executive Secretary, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 13½ hours of category 1 credit.

Seventh Annual Frontiers in Ophthalmology
February 12-14, 1981. The Arizona Biltmore Resort. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator, St. Luke's Hospital. 525 N. 18th St., Phoenix, Arizona (602) 258-7373, Ext. 311. Approved for 15 hours of category 1 credit.

Third Annual Practical Counseling Technique Workshop for the Primary Physician
February 14-21, 1981. Lion Square Lodge, Vail, Colorado. Sponsors: Institute for Human Relationship and Development and Colorado Academy of Family Physicians. Contact: John C. Faul, M.D., 5290 E. Yale Circle, Ste 200, Denver, Co. 80222.

Third Annual International Catarct Surgery Symposium
February 16-18, 1981. La Posada Resort Hotel, Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

Ninth Annual Taos Lung Disease Symposium
February 20-22, 1981. Holiday Inn, Taos, New Mexico. Sponsor: New Mexico Chapter of the American Thoracic Society. Contact: New Mexico Chapter of the American Thoracic Society, 216 Truman, NE, Albuquerque, New Mexico 87108.

PEDIATRIC UPDATE - 1981

Sponsor:	Department of Pediatrics Long Island Jewish-Hillside Medical Center New Hyde Park, N.Y.
Location:	Marbella, Spain, on the Costa del Sol
Dates:	February 28-March 8, 198
Credits:	20 credits AMA, Category 1 20 credit LCCME
Faculty:	International faculty includes: Professor Victor Dubowit London, England Professor John O. Forfar Edinburgh, Scotland Professor Walter H. Hitzi Zurich, Switzerland Professor Ettore Rossi Bern, Switzerland Professor R. Zetterstrom Stockholm, Sweden
Tuition:	$185; $315 MAP (full American breakfast and dinner)
Information	**Continuing Education Coordinator** **Long Island Jewish-Hillside Medical Center** **New Hyde Park, N.Y. 1104** **(212) 470-2114**

EKG Interpretation and Arrhythm Management
February 20-21, 1981. Hyatt Regency Hote Phoenix, AZ. Sponsor: International Medic Education Corp. Contact: Stephen Mattingl Vice President, International Medic Education Corp., 64 Inverness Dr. Eas Englewood, Co. 80112. Approved for hours of category 1 credit.

Fourth Annual Mid-Winter Symposium OB/GYN
February 25-27, 1981. Scottsdale Hilto Hotel, Scottsdale, Arizona. Sponso Maricopa County General Hospital, Dept. OB/GYN, and Phoenix OB/GYN Societ Contact: Secretary, Phoenix OB/GY Society, 333 E. Virginia, Ste 222, Phoenix, A 85004. (602) 257-9218.

Personal Instruction Course in Catara Surgery
February 25-28, 1981. The Eye Foundatio Phoenix, AZ. Sponsor: The Eye Foundatio Contact: The Eye Foundation, 421 N. 18t Street, Suite 105, Phoenix, AZ. Approved f hour per hour category 1 credit.

Prophlactic Antibiotics in Surgery
February 27, 1981. Boswell Memori Hospital, Sun City, AZ. Sponsor and Contac Office of CME, Walter O. Boswell Memori Hospital, 10401 Thunderbird , Sun City, A Approved for hour per hour category 1 credi

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TIERS IN

OPHTHALMOLOGY

1981

The seventh annual **Frontiers in Ophthalmology** seminar will explore the latest research and techniques in significant areas of ophthalmologic care. A distinguished faculty has been selected to conduct the seminar sessions.

Topics will include:
Corneal and Anterior Segment Surgery • Ocular Plastic Surgery • Strabismus • Retinal and Vitreous Surgery • Neuro-Ophthalmology • Diagnosis and Treatment of Corneal Disease • and more.

to be held at the world famous 5-star
Arizona Biltmore Hotel
Phoenix, Arizona
February 12 - 14, 1981

This seminar meets the criteria for 15 hours of C.M.E. credit.

Sponsored by the Prentice Eye Institute of St. Luke's Hospital Medical Center

For additional information, write or call:
Christine Campbell, Seminar Coordinator/St. Luke's Hospital Medical Center
525 N 18th Street, Phoenix, Arizona 85006/(602) 258-7373 ext. 311

14th Annual Southwestern Clinical Pharmacy
Clinical Aspects of Psychotherpauetics
February 27-March 1, 1981. Ramada Inn, Tucson, AZ. Sponsor: U of A College of Pharmacy. Contact: Jack R. Arndt, Ph.D., College of Pharmacy, U of A, Tuson, AZ 85721. Approved for 8 hours of category 1 credit.

MARCH

Ski CME Program
March 21-26, 1981. Vail, Colorado. Sponsor: Desert Samaritan Hospital. Contact: L. A. Rosati, M.D., CME Committee, Desert Samaritan Hospital, 1400 South Dobson Road, Mesa, Arizona. Approved for 16 hours of category 1 credit.

Crisis and Emergency Pediatrics
March 5-8, 1981. Doubletree Inn, Tucson, AZ. Sponsor: U of A College of Medicine, and Department of Pediatrics. Contact: Hugh C. Thompson, M.D., Pediatrics, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 17 hours of Category 1 credit.

Annual Meeting—Society of Head and Neck Surgeons
The American Society For Head and Neck Surgery
March 8-11, 1981. The Hyatt Regency Hotel, Phoenix, Arizona. Sponsor: American Society of Head and Neck Surgery. Contact: Gabriel F. de Freitas, M.D., Locall Arrangements Chairman, Suite 101, 1901 E. Thomas Road, Phoenix, AZ 85016.

Personal Instruction Course in Catract Surgery
March 11-14 and 25-28. The Eye Foundation, Phoenix, Az. Sponsor and Contact: The Eye Foundation, 421 n. 18th Street, Suite 105, Phoenix, AZ 85006. Approved for hour per hour category 1 credit.

Seventh Annual Maricopa County General Hospital
Urological Seminar
March 14 & 15, 1981. The Registry Resort, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: James H. McDonald, M.D., Chief of Urology Service, MCGH, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 14 hours of category 1 credit.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.

Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M.. J-6 Conf. Rm., Contact: Dr. Conger & Staff

Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.

Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgical Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third and 5th, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists

Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.

Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.

Tumor Board
Last Thursday of the month, 12 Noon, Educ. Center Classroom I & II, May, July, Sept, & Nov.

Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II. Contact: Brian Updegraff, M.D.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: T. F. Reid, Jr., M.D.

Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.

CME Programs
Weekly Tuesday, 7 p.m., Cafeteria-West End.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.

OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

DOCTORS HOSPITAL

1747 East Thomas Road, Phoenix, Arizona 86016 Contact: Medical Staff Secretary for additional information.

Physicians Continuing Educaiton Program
1st Thursday, 12:30 p.m., Classrooms.

ELDORADO HOSPITAL TUCSON (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: W. J. Howard, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.

Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm

Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.

Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.

Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.

Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North

Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam Practice Conf. Rm.

Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 A.M., Contact: Paul Bergeson, M.D.

Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.

Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm Contact: John A. Bruner, M.D.

Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.

Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm

Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.

OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm

PM & R Grand Rounds
2st. & 4rd. Wed., 12:00-1:00 P.M., R-2 Conf Rm.

Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf Rm.

GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151 Radiology Conf. Rm.

Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr Ben Ora

Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.

Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology

Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.

Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm

Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.

Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.

Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.

PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: J. Goldberger, M.D.

Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman

Ophthalmology Conference
1st Monday, 12:30 P.M., Contact: Bruce Gillers, M.D.

Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.

Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D.

Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: B. Phibbs, M.D.

Clinical Pharmacology Conference
5th Tuesday, 12:30 P.M., Contact: M. Katz, Clinical Pharm.

12Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact J. Laguna, M.D.

Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: M. Baker, M.D.

Medical Surgical Grand Rounds
2nd Wed., 12:30 P.M., Contact: Chief Medical Resident

Medical Conference
1st Wed, 12:30 P.M., Contact: Chief Medical Resident

Cardiology Conference
1st Thursday, 12:30 P.M., Contact: B. Phibbs, M.D.

Endocrine Conference
2nd Thursday, 12:30 P.M., Contact E. Munjack, M.D.

GI Conference
4th Thursday, 12:30 P.M., Contact. C. Kanun, M.D.

Mortality & Morbidity Conference
3rd. Thursday, 12:30 P.M., Contact: E. Munjack, M.D.

OB/GYN Dept. Meeting
2nd Thursday, 2 P.M., Contact J. Wurzel, M.D.

OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: J Wurzel, M.D.

OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 P.M., Contact: J. Wurzel, M.D.

Clinical Pathology
5th Thursday, 12:30 P.M., Contact: J. Fleishman, M.D.

Renal Conference
1st Friday, 12:30 P.M., Contact: T. Pence, M.D.

Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D.

Rheumatology Conference
3rd Friday, 12:30 P.M., Cntact J. Goldberger, M.D.

Chest Conference
5th Friday, 12:30 P.M., Contct: D. Engelsberg, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008
Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

Clinical Conferences
2nd & 4th Wednesday, 12-1 p.m., Medical Staff Lounge

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 85008. Contact: Leonard Tamsky, M.D.

Medicine Conference
Daily 12-1 P.M., Contact: S. Schaffner, M.D.

Chest Surgery Conference
Weekly, Mondays, 1:30 P.M., Santa Cruz Rm.

Hematology Conference
2nd Tuesday, 1:00 P.M., Contact: Walt Koppenbrink, M.D.

OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Room

Obstetrical Problem Conference
Weekly Tuesday, 7:30 A.M., Yavapai Rm.

Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.

Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.,Contact: Robert Ganelin, M.D.

Urology Discharge Planning Conference
Weekly, 11:30 A.M., Station 42

Hand Surgery Conference
Weekly Wed, A.M., Santa Cruz Rm.

Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.

OB/Neonatology Seminar
Weekly, Wed. 7:30 A.M., Yavapai Rm.

Clinical Psychiatric Conference
Weekly, Wed., 11-12 P.M. Mental Health Annex, Rm. 1346.

Surgery Conference
Weekly Wed., 7-8 A.M., Surgical Dept.

Current Concepts in Medicine & Surgery
1st Thursday, 1 p.m., Dr. Hospital Class Rm., Contact: Dr. Tamsky.

Cardiology Conference
Weekly Thurs., 2 P.M., Santa Cruz Rm.

OB/GYN Resident Conference
Weekly Thurs., 12 P.M., Yavapai Rm.

GYN Endocrine Seminar
1st & 3rd Friday, 12:30 P.M., Sanata Cruz Rm.

OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 A.M., Yavapai Rm.

Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & HEALTH CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, Wed. and Friday, 12 Noon

OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conf. Rm.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for Category 1 credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Contact: D. Randall, M.D.

Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 A.M.

Otolaryngology Grand Rounds
4th Wednesday, 4-5 p.m., Conference Rm. A, Contact. N. Wendell Tood, M.D.

Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 A.M., Conf. Rm A, Contact. Drs. L. Fairbanks & E.Y. Hooper.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact. George Scharf, M.D. Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm

Clinical Conferences
Weekly Tuesday, 12 Noon, Kiva Conference Room

Tumor Board Conference
Weekly Friday, 12 P.M., Kiva Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:30 P.M., Doctors' Lounge

Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge

Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge

Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 P.M., Doctors' Lounge

Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge

Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge

Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge

Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge

Cardiolpulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge

OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge

Trauma Conference
3rd Thurs, 12:30 P.M., Doctors' Lounge

Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge

Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge

GI Medical—Surgical Rdiological Conf.
2nd Friday, 12:30 P.M., Doctors' Lounge

Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.

Genetics Conference
Weekly Monday, 12:30 P.M., Pediatric Department.

Pediatric Rounds
Weekly Monday, Wed. & Friday, 10:30 A.M., Pediatric Department.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.
ECG Conference
Weekly Tuesday, 12:30 P.M., Pediatric Department.
Medical Grand Rounds
Weekly Wednesday, 8 A.M., 1st Floor Conf. Room.
Visiting Professor Formal Presentation
Weekly Thursday, 8 A.M., PIMC
Visiting Professor Informal Presentation
Weekly Thursday, 9:30 A.M., 1st floor Conf. Rm.
Visiting Professor Formal Presentation
Weekly Thursday, 12:30 P.M., PIMC
Nephrology Conference
Weekly Fridaym 12:30 P.M., Pediatric Department.

ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N. Wilmot Road, Tucson, AZ. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Family Practice Department Meeting
3rd Tuesday, 12 P.M., Contact: Wm. Moteforte.
Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 P.M., Contact: Kim Sowards.
Current Concepts in Medicine
Weekly, Tuesday, 12 P.M., Auditorium.
Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 P.M., Contact: S. Salmon, M.D.

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.

Cardiac Conference
Weekly Monday, 12:15 P.M., Auditorium
Surgery Conference
1st Tuesday, 12:15 P.M., Auditorium
Emergency Medicine Conference
1st Wednesday, 12:15 P.M., Auditorium
Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 P.M., Auditorium
Psychiatry Conference
3rd Thursday, 7 A.M., Auditorium
Combined Medical General Practice Conference
1st Friday, 12:15 p.m., Auditorium
Toxicology Grand Rounds
2nd Friday, 7:30 A.M., Auditorium
Ophthalmology Conference
1st Saturday, 8:30 A.M., Auditorium.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Emergency Medicine Lectures
Weekly Thursday, 8 A.M., Century Room A
Mental Health Update
1st Friday, 11:30-1:00 P.M., Century Room A
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Cardiology Conference
Weekly Monday, except last, 12 Noon, Contact: J. Fernadez, M.D.
Dermatology Conference
4th Monday, 5:00 P.M., Contact: R. Miller, M.D.
Endocrinology Conference
Last Monday, 12 P.M., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.
Chest Conference
3rd Tuesday, 12 Noon, Contact: William Nevin, M.D.
Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Infectious Disease Conference
2nd Tuesday, 12 Noon, Contact: B. Friedman, M.D.
Nephrology Conference
1st Tuesday, 12 Noon, Contact: Dr. Seltzer.
Orthopedic Conference
1st Tuesday, 8:00 P.M., Contact: Jay Katz, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 P.M., Contact: Dr. Lightner.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.
Clinical Pathology Conference
Last Wednesday, 8:00 A.M., Contact: Dr. Fuchs.
Family Practice Meeting
2nd Wednesday, 12:30 P.M., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.
Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buschsbaum, M.D.
Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.
Tumor Conference
Weekly Thursday, 12 Noon, Contact: Dr. Hastings.
G—I Conference
Weekly Friday, 12 Noon, Contact: C. Krone, M.D.
Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.
OBGYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.
OBGYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.
Surgical Department Confernece
Weekly Saturday, 7:30 A.M., Contact: Dr. Hastings, TMC.
Surgical Grand Rounds
Weekly Saturday, 9:00 A.M., UAHSC-Room 5403, Contact: Dr. Norton.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.
Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact: Drs. Otto & Zehngut.
Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 P.M., HSC Auditorium, Contact: Cancer Center
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Room 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.
Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn
Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern



Acute pain is no laughing matter.

The first prescription for the first days of acute pain

Empirin® c̄ Codeine #3

Each tablet contains: aspirin, 325 mg; plus codeine phosphate, 30 mg, (Warning — may be habit-forming). Ⓒ III

For the millions of patients who need the potency of aspirin and codeine for their acute pain.

The pain of fractures, strains, sprains, burns and wounds is at its peak during the first three to four days following trauma. The potent action of Empirin c̄ Codeine begins to work within 15 minutes of oral administration, an important advantage during this acute pain period. Empirin c̄ Codeine has unique bi-level action to attack pain at two critical points: peripherally at the site of injury and centrally at the site of pain awareness.

For the most effective dosage in treating acute pain, begin with... two tablets of Empirin c̄ Codeine #2 or #3, every four hours. Titrate downward as pain subsides.

EMPIRIN® with Codeine

Ⓒ III

DESCRIPTION: Each tablet contains aspirin (acetylsalicylic acid) 325 mg plus codeine phosphate in one of the following strengths: No. 2 — 15 mg, No. 3 — 30 mg, and No. 4 — 60 mg. (Warning — may be habit-forming.)

CONTRAINDICATIONS: Hypersensitivity to aspirin or codeine.

WARNINGS:

Drug dependence: Empirin with Codeine can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of this drug and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral, narcotic-containing medications. Like other narcotic-containing medications, the drug is subject to the Federal Controlled Substances Act.

Use in ambulatory patients: Empirin with Codeine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. The patient using this drug should be cautioned accordingly.

Interaction with other central nervous system (CNS) depressants: Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, other tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) concomitantly with Empirin with Codeine may exhibit an additive CNS depression. When such combined therapy is contemplated, the dose of one or both agents should be reduced.

Use in pregnancy: Safe use in pregnancy has not been established relative to possible adverse effects on fetal development. Therefore, Empirin with Codeine should not be used in pregnant women unless, in the judgment of the physician, the potential benefits outweigh the possible hazards.

PRECAUTIONS:

Head injury and increased intracranial pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal conditions: The administration of Empirin with Codeine or other narcotics may obscure the diagnosis or clinical course in patients with acute abdominal conditions.

Allergic: Precautions should be taken in administering salicylates to persons with known allergies. Patients with nasal polyps are more likely to be hypersensitive to aspirin.

Special risk patients: Empirin with Codeine should be given with caution to certain patients such as the elderly or debilitated, and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture, peptic ulcer, or coagulation disorders.

ADVERSE REACTIONS: The most frequently observed adverse reactions to codeine include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include euphoria, dysphoria, constipation, and pruritus.

The most frequently observed reactions to aspirin include headache, vertigo, ringing in the ears, mental confusion, drowsiness, sweating, thirst, nausea, and vomiting. Occasional patients experience gastric irritation and bleeding with aspirin. Some patients are unable to take salicylates without developing nausea and vomiting. Hypersensitivity may be manifested by a skin rash or even an anaphylactic reaction. With these exceptions, most of the side effects occur after repeated administration of large doses.

DOSAGE AND ADMINISTRATION: Dosage should be adjusted according to the severity of the pain and the response of the patient. It may occasionally be necessary to exceed the usual dosage recommended below in cases of more severe pain or in those patients who have become tolerant to the analgesic effect of narcotics. Empirin with Codeine is given orally. The usual adult dose for Empirin with Codeine No. 2 and No. 3 is one or two tablets every four hours as required. The usual adult dose for Empirin with Codeine No. 4 is one tablet every four hours as required.

DRUG INTERACTIONS: The CNS depressant effects of Empirin with Codeine may be additive with that of other CNS depressants. See WARNINGS.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.
Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson
Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.
Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford
Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room
Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross
Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.
Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley
Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.
Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.
Psychiatric Grand Rounds
Weekly Wednesday, 5:30 P.M., AHSC, Rm 8403, 5th Floor Auditorium
Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson, Palo Verde Hospital
Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.
Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.
Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson
Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655
Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich
Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.
Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C.
Uroradiology (Genitourinary Radiology)
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology
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1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.
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Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.
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Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 P.M., RM. 3134, Contact: Dr. Mertz, Ext 493.
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GI Grand Rounds
Weekly Tuesday, 1 P.M., Contact: Drs. Sanowski, Kozarek & Partyka
Cancer Symposium
Weekly Wed., 1 P.M., T-5, Contact: Dr. Byrne, Ext. 426.
Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 P.M., Contact: Dr. Rohwedder, Ext. 388.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 P.M., Rm 3134. Contact: Dr. Mertz, Ext. 493.
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Cardiology Conference
2nd Thursday, 1 P.M., Rm. T-5.
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Weekly Wed., 12-1:00 P.M., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam
Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.
Grand Rounds
Weekly, Thursday, 11 A.M., Bldg. 107, Contact J. Fitzharris, D.O.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon.
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ArMA/AMA UNIFIED MEMBERSHIP REQUIREMENT DISSOLVED

THE HOUSE OF DELEGATES OF ArMA IN A SPECIAL MEETING ON NOVEMBER 1ST VOTED overwhelmingly (106 YES to 20 NO) to adopt Resolution S-1-80 which changes the Association's bylaws so that membership in the American Medical Association is voluntary.

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The issue of Unified membership with the AMA has been debated at seven House of Delegates meetings during the past five years, but, until last Saturday's meeting, the proponents of voluntary membership with the AMA have not been able to muster the two-thirds vote necessary to change the bylaws.

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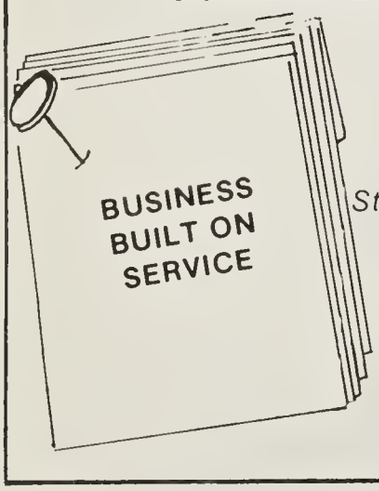


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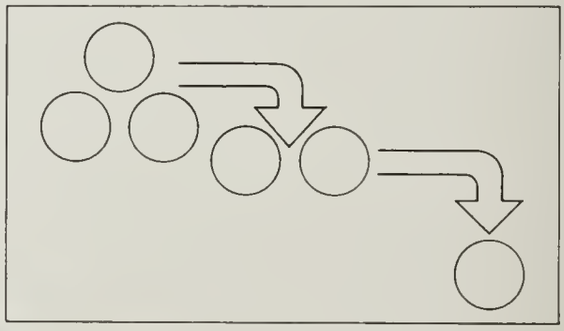
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*Sellers EM: *Drug Metab Rev* 8(1):5-11, 1978



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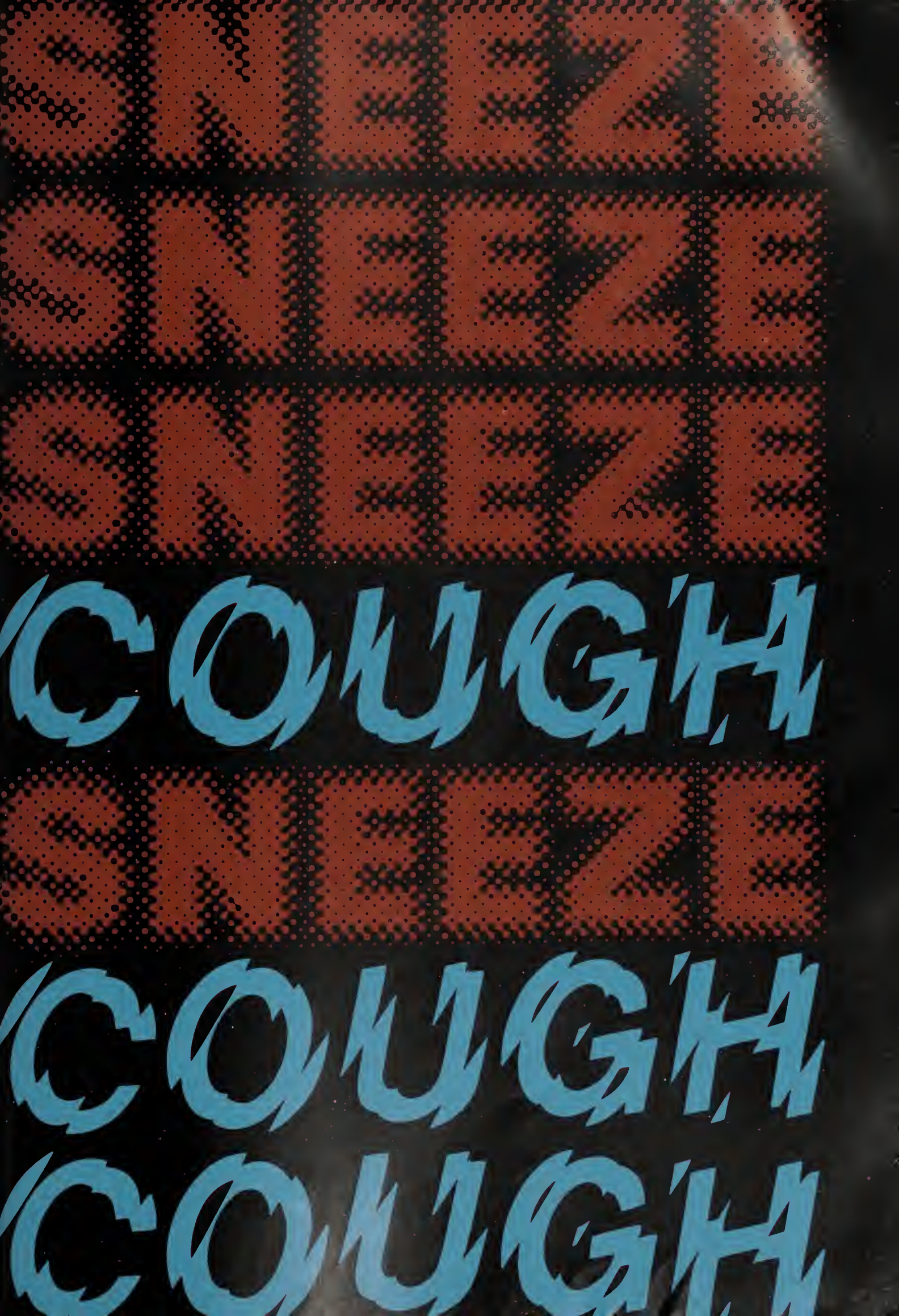
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WARNINGS Ru-Tuss Tablets may cause drowsiness Patients should be warned of the possible additive effects caused by taking antihistamines with alcohol, hypnotics, sedatives or tranquilizers
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DOSAGE AND ADMINISTRATION Adults and children over 12 years of age one tablet morning and evening Not recommended for children under 12 years of age Tablets are to be swallowed whole
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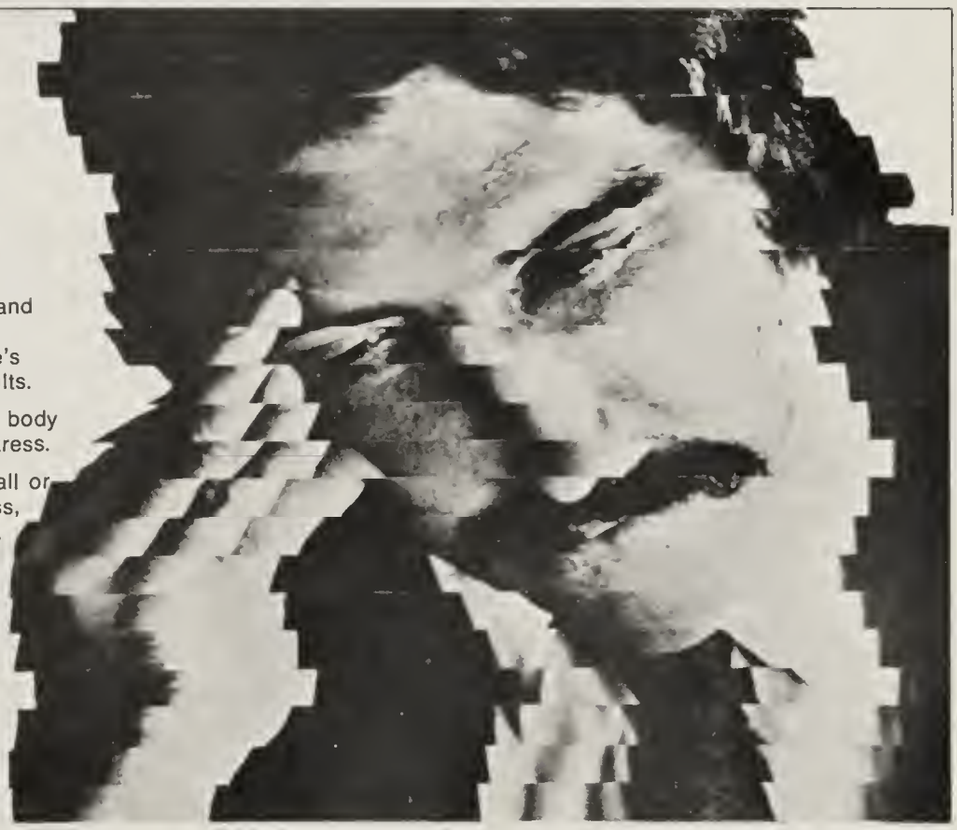
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Pruritus as a Sign of Systemic Disease

Report of Metastatic Small Cell Carcinoma

Daniel E. Beeaff, M.D.

Editors:
Ronald C. Hansen, M.D.
Robert A. Schwartz, M.D.

Introduction

Pruritus is a common problem which is usually a temporary annoyance. Occasionally the pruritus can be so severe that it requires medical attention. Often it can readily be attributed to an obvious skin condition such as atopic dermatitis or scabies. However, in those cases of generalized pruritus without evidence of a primary cutaneous disorder, one should consider the possibility of an underlying internal disorder. The following case description will illustrate that association.

Case Report

The patient was a 51-year-old white male. His daughter and her children had been seen earlier for a typical case of scabies they and the immediate family, including the patient, were treated with crotamiton cream 10%. The daughter and her children cleared completely with therapy, however, the patient continued to itch. On his initial visit he complained of generalized, intense pruritus for the preceding 8 weeks. He had come in contact with his daughter and the grandchildren multiple times during that period. He associated the itching with some personal problems and found the itching worse when worrying at night. The crotamiton cream did not help him. His general health had been excellent with the exception of some low back problems which were bing treated by his local physician. Physical exam was unremarkable with the exception of multiple excoriations randomly scattered over his trunk and extremities, particularly the lower legs. Neurodermatitis and scabies were the leading considerations. He was treated with triamcinolone cream 0.1% T.I.D. and hydroxyzine hydrochloride 25 mg p.o. Q.I.D. Over two weeks there was slight improvement in the itching, but again he associated it with increasing personal stresses. Neurodermatitis aggravated by his anxieties remained the working diagnosis. His treatment was altered to Shepard Lotions with 0.025% menthol and 3% liquor carbonis detergent, and chlordiazepoxide hydrochloride 10 mg p.o. T.I.D. An SMAC 20 showed the following abnormalities: Alkaline phosphatase of 294 U/l (Normal = 30-115), SGOT 62 U/l (Normal = 0-41), SGPT 45 U/l (Normal = 0-45), and LDH 1247 U/l (Normal = 60-200). The patient returned 2 weeks later on his third visit complaining of easy bruising. Physical examination showed multiple areas of purpura, primarily ecchymoses, scattered over his entire body. In addition, there were several discrete erythematous to purpuric papules, some perifollicular and some excoriated, over the anterior shins. (Figure 1). A stat blood count showed the following abnormalities: hematocrit 30, hemoglobin 10.2, white cells 9,900 with neutrophiles 37%, lymphocytes 45%, Stabs 13% and nulceated RBS's 98%, platelet count of 60,000 and a normal prothrombin and partial thromboplastin times. A bone marrow examination demonstrated a metastatic, undifferentiated small cell carcinoma, with oatcell carcinoma of the lung the primary consideration. A chest x-ray with apical lordotic view suggested a left-hilar soft-tissue nodule. However, bronchoscopy with biopsy and cytology were negative. Skull series showed lytic lesions within the skull and an EMI scan of the head suggested metastatic disease in the right parietal bone. Spinal x-ray showed a compression fracture of the T7 vertebra. The patient received one course of Vincristine 2 mg, Andriamycin 60 mg, and Cytoxan 1 gm while in the hosptial, and was discharged to follow-up in Oncology clinic. Approximately two weeks later the patient was readmitted with congestive heart failure, possibly secondary to a myocardial infarction and aggravated by his anemia. His pruritus had spontaneously resolved. He was discharged again to follow-up as an outpatient.

Discussion

This case illustrates the ease with which pruritus can be wrongly attributed to something as common as scabies or to emotions. One could still maintain that scabies best accounted for the itching, but his subsequent course is strongly against this diagnosis. The absence of definite primary skin lesions, and the presence of only excoriations should alert the physician

From: 601 North Wilmot Road, Tucson, Arizona 85011



Figure 1
Clinical photographs of patient on hospitalization demonstrating the purpura, and erythematous, purpuric papules with excoriations, over the shins.

Table 1
Systemic Diseases Associated with Generalized Pruritus

1. **Metabolic**
 a. Thyroid disease: Hyper- and Hypothyroidism
 b. Hypoparathyroidism
 c. Diabetes Mellitus (?)
 d. Carcinoid Syndrome
 e. Gout (?)
2. **Hepatic**
 a. Cholestatic Liver Disease
 b. 1° Biliary Cirrhosis
3. **Renal**
 a. Uremic Pruritus
4. **Hematological**
 a. Polycythemia Vera Rubra
 b. Myelofibrosis
 c. Paraproteinemia
 d. Mast Cell Disease
 e. Leukemia
 f. Lymphoma
 g. Myeloma
5. ***Visceral Malignancy**
6. **Miscellaneous**
 a. Pregnancy
 b. Drug induced
 c. Parasitic

that an underlying disorder must be considered. Not until the patient presented with purpura did we consider malignancy as the cause of his pruritus. It should be recalled that generalized pruritus can be a presenting sign of systemic disease and that a limited workup must be considered before attributing the itching to nerves or neurodermatitis.

Approximately $11 million dollars is spent annually for itch suppressants, and, if one includes the cost of topical steroids at $100 million per year, of which a large portion is prescribed for pruritic disorders, then one can get some idea of the frequency and cost of pruritus.[1] Many of the causes of pruritus occur with primary cutaneous disease, such as scabies, dry skin, atopic eczema, contact dermatitis or dermatitis herpetiformis and one can usually find evidence for these diseases in physical exam. In those cases where no primary skin disorder is found, and there is generalized pruritus with only secondary excoriations it has been estimated that from 20%-30% may be found to harbor an underlying systemic disease[1] (Table 1). Of those systemic diseases, the pruritus may accompany an underlying endocrinopathy, liver, renal or hematological disease and as in this case a visceral malignancy.

Association with visceral malignancies has been infrequently reported. Cases where pruritus antedated the malignancy and cleared when the malignancy was treated have been described for: lymphoepithelioma of the nasopharanyx,[2] prostatic carcinoma,[2,9] stomach

arcinoma,[2] adenocarcinoma of the breast,[2] metastatic denocarcinoma of the stomach,[4] bronchogenic arcinoma,[5,9] uterine carcinoma,[6] metastatic denocarcinoma to the scapula,[3] metastatic thyroid arcinoma,[9] and pyloric stomach carcinoma.[7] Cormia[2] lescribed his cases as presenting with intractable pruritus prior to the diagnosis of the malignancy. The ites of predominant involvement were the trunk, xtremities, particularly the anterior surfaces of the ower legs and ankles. The itching was generalized with ntense burning sensations and did not respond to any opical therapy. The lower legs frequently showed small papules he described as 'scratch papules.' The patients most often were initially asymtomatic for their underlying tumor and it was found only by a workup for an occult process. The pruritus in most cases cleared spontaneously when the malignancy was treated. In our case, the patient complained of an intense generalized pruritus with excoriations and papules over the shins, almost two months before the diagnosis of an underlying malignancy. His pruritus was generalized and had a burning quality. The pruritus resolved following the initiation of therapy for metastatic undifferentiated small cell carcinoma.

The proposed mechanisms for pruritus are more complex than this disucssion would permit and the reader is referred to reviews by Herndon[8] and Lyell.[5] Evidence has shown that pruritus, or that sensation which evokes the response of scratching, is mediated by histamine, kinins, prostaglandins, endopeptidases, cathepsins, that may directly act on itch nerve receptors present at the dermoepidermal junction and possibly just within the epidermis. The older theories of VonFrey with the concept that skin sensation was served by specific sites, such as a site for warmth, cold and so forth and that there were specific structures mediating different sensations, has been replaced by a physiological rather than anatomical theory referred to as the Gate Control Theory of Melzack and Wall. The above references discuss these various aspects of pruritus in much more detail. The mechanisms of itching in visceral malignancies has not been adequately explained. Rothman[10] felt that there were small microscopic metastatic implants of the tumor occurring in the skin and that itching was occurring because of immune response to these tumor cells. Other thoughts have been that the tumor is producing some chemical mediator that is producing the itch. In some cases the itching may have been simply coincidental with the malignancy. Those cases, as this one, in which the itching resolves with therapy of the malignancy, does raise the question of a true relationship to the malignancy.

How far one works up such a patient depends on several factors. Certainly a very basic workup with chemistry profile, including liver function tests, total blood count, urinalysis, VDRL, stool of O and P, skin scrapings for scabies, skin biopsy plus immunofluorescense and chest x-ray should be considered. More specific tests can be ordered for specific findings, problems or complaints. Because of the common nature of this problem, some discretion is needed to avoid an unnecessarily expensive evaluation. Lyell[5] found a significant internal disorder in only 5 of 74 patients (6%) with generalized pruritus. These disorders included: two with renal disease, two with cancer and one with liver disease. Rajka[9] found 16 of 34 patients (47%) with an underlying disorder. His series included six reticuloses, four renal diseases, three cancers, two liver diseases and one diabetes mellitus. In spite of such widely different percentages one needs to keep in mind the possibility of potentially serious internal disorders as a cause of unexplained pruritus. Approaching the otherwise healthy appearing patient conservatively is certainly worth a trial. This may involve simply correcting aggravating environmental factors such as excessive bathing, hot water, harsh soaps, low room humidity, topical irritants, and inadequate use of emollients. If the pruritus intensifies or remains unrelieved then further evaluation should be considered.

Pruritus can be symptomatically managed with bland lotions containing one-fourth percent menthol and one-half percent phenol or with 3-6% liquor carbonis detergents. Topical steroids are sometimes helpful in addition oral antihistamines can be used. In the pruritus of liver disease cholestyramine and phenobarbital have been found useful, and in uremic pruritus, increasing doses of UVB irradiation can be highly effective. Suffice it to say that the definitive therapy should be directed toward the underlying disorder. As in our patient, nothing helped the patient's pruritus until therapy for his malignancy was started, thus strengthening the case for causation by his malignancy.

Acknowledgements

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Seminars in Gastroenterology and Liver Disease

Gastritis

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A very common diagnosis made by physicians who treat patients with abdominal pain is gastritis. Often, an upper gastrointestinal series is done which shows mucosal fold thickening in the absence of an obvious ulcer. More often, however, the UGI series is normal in this condition.

Gastritis is a condition which is diagnosed most accurately by histologic methods. The two broad classifications are that of actue and chronic gastritis. In either group, the histologic abnormality may be diffuse, involving most or all of the stomach, or localized or patchy.

Acute Gastritis

The histology of acute gastritis is that of varying degee of inflammation with polymorphonuclear leukocytes located in the lamina propria, associated with edema and capillary engorgement. The surface epithelium may be eroded away, and bleeding can ensue. The causes are numerous. (Table 1).

In all instances, the correlation between symptoms and degree of inflammation are not equivalent, with the symptoms usually being less marked than the histologic changes. Symptoms include epigastric discomfort, often of a burning nature, nausea, vomiting, gaseousness and bloating. The treatment consists primarily of removing the offending agent, which often results in prompt symptomatic improvement. Antacids, simethicone, antispasmodics and cimetidine can provide relief of symptoms.

Table 1
Causes of Acute Gastritis

Drugs (Salicylates, nonsteroidal anti-inflammatory agents, colchicine, quinine, caffeine, iodine)
Alcohol
Malnutrition
Emotion (Anxiety)
Bacterial Toxins (polio, diphtheria, influenza, salmonella staphylococcus)
Irradiation
Food Overindulgence (Volume and spices)
Corrosives (Mercury, acid, nasogastric tube
Mechanical Trauma
Disease (uremia, pulmonary, cirrhosis, Neurologic)

Acute hemorrhagic gastritis is an often overwhelming disorder characterized by sudden gastrointestinal hemorrhage. There is usually no pain or other premonitory symptoms. It may occur in the setting of another severe illness, such as burns, CNS disease, uremia, or severe pulmonary disorders, and is also referred to as stress ulcer. It can also be associated with a sudden acute anxiety, or after the use of gastric mucosal irritating agents, such as alcohol, aspirin, and the nonsteroidal, anti-inflammatory drugs. The diagnosis is made best by upper intestinal endoscopy, and a severely inflammed, oozing, eroded mucosa is seen, at times with multiple shallow ulcerations apparent. These tend to be present more commonly in the proximal part of the stomach, although the antrum is more likely to be involved if aspirin or drugs are the cause. Treatment is begun with gastric lavage, and antacids are administered in an amount sufficient to keep the intragastric pH above five. Intravenous cimetidine is a very useful adjunct in this condition, and either this drug or antacids have been promulgated as effective prophylactic measures for patients with serious conditions likely to be complicated by stress ulcer bleeding. Eighty to ninety percent of such bleeding can be controlled by medical measures,[2] but occasionally surgical intervention becomes mandatory. This is particularly dangerous in a patient who may have another serious life-threatening condition.

A fortunately rare disorder is **phlegmonous gastritis**, in which bacteria invade the gastric wall. It is seen in a

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etting of trauma, hypoacidity, chronic malnutrition, lcoholism, or infection elsewhere in the body. The atient is acutely ill with pyrexia, nausea, vomiting, evere abdominal pain, and peritoneal signs. The tomach is dilated and may show a linitis plastica-like icture on UGI series. Leukocytosis is nearly always resent. The organism is frequently a gram positive acterium, and in about half of the cases is a treptococcus. Antibiotics lead to defervescence, but urgical intervention is necessary to prevent mortality. The seriousness of this disorder is shown by the nortality which may exceed 50%.[2]

Chronic Gastritis

The more common form of gastritis is the chronic ariety, which has been further sub-classified as uperficial, atrophic and hypertrophic. In *superficial astritis*, the lamina propria contains lymphocytes, lasma cells and eosinophils, usually confined to the uter third of this layer. In *atrophic gastritis*, the nflammatory elements are the same as in superficial astritis, but is more extensive. Grandular size is decreased and there is loss of parietal and chief cells. There is increased space between the glands, and these may be variably absent, resulting in the mucosal thinning characteristic of the more advanced stage of this condition. In the most advanced degree, the remaining cells become columnar, develop goblet cells and a brush border, and can assume an absorptive function. This is referred to as intestinal metaplasia. A malignant potential is associated with this finding.

Superficial gastritis can persist, revert to normal, or progress to atrophic gastritis. Atrophic gastritis is associated with the development of adenocarcinoma in up to 10% over many years.

The cause of these conditions is unclear, but genetic influences, iron deficiency, endocrine and immunologic factors have been incriminated. The latter have been extensively studied. Parietal cell antibodies develop in many patients with atrophic gastritis, and are associated with pernicious anemia. These occur in 90% of patients who develop pernicious anemia, but are also seen in about 50% of patients with atrophic gastritis who do not develop pernicious anemia. Two types of antibodies are also found to intrinsic factor, a blocking antibody which interferes with the combination of Vitamin B_{12} and intrisic factor (type I), and a binding antibody which prevents adherence of the B_{12} - IF complex to the absorptive site in the terminal ileum (type II).[3] Antibodies have also been demonstrated to the gastrin producing cells of the antrum, but in less than 10% of patients.[4]

Most patients with atrophic gastritis do not develop pernicious anemia. In part, this may be due to the patchy distribution of the lesion, resulting in sufficient production of intrinsic factor to prevent the development of pernicious anemia. For the same reason, these patients may not be achlorhydric, as acid and intrinsic factor are both produced in the parietal cell. Some patients have sparing of the antrum by the inflammatory process, whereas in others the entire stomach is involved. This has resulted in the designation of type A and B chronic gastritis.[4] In type A chronic gastritis, the antrum is spared, and the serum gastrin level is elevated. These patients have parietal cell antibodies and often intrinsic factor antibodies, are achlorhydric, have latent or overt pernicious anemia, and have a high association with other autoimmune endocrine disorders. In type B chronic gastritis, the antral mucosa is primarily affected, parietal cell antibodies may not be present, and intrinsic factor antibodies are absent. Pernicious anemia does not develop. The serum gastrin is normal or low, and hypo- or achlorhydria occurs. Type B chronic gastritis is more common than type A, and is also more likely to be associated with the symptoms of indigestion, upper abdominal pain, anorexia and vomiting. The development of chronic gastric ulcer and upper GI bleeding is also more common in the type B form.[5]

The treatment of this disorder is symptomatic, but as chronic gastritis often results in no complaints from the patient, no specific therapy need be offered. The administration of hydrochloric acid is usually not helpful. Vitamin B_{12} injections are needed life-long in patients who develop pernicious anemia. A monthly injection of 1 mg is usually adequate.

Hypertrophic gastritis is a condition in which there is an increase in the thickness of the mucosa of the stomach, caused by enlargement of the glands, They are enlongated, giving a cork-screw-like appearance. In some areas the glands degenerate and become cystic. The lamina propria is infiltrated with plasma cells, polys, lymphocytes and eosinophils. Edema is present. Grossly, the folds are very much enlarged, up to 3 - 4 cm in size, and may produce a nodular, cobble-stone or tumor pattern on x-ray. The antrum is usually spared. Acid levels may be diminished as a result of neutralization and dilution by the large amounts of mucus and fluid present. A severe protein loss may occur. This is known as Menetrier's disease. Symptoms include epigastric pain, anorexia, nausea, vomiting and bleeding. Edema and malnutrition occur as protein loss may be large and cannot be corrected by oral intake. Gastrectomy may be necessary if protein intake, antacids and cimetidine do not control the defect.

An analogous condition called hyertrophic, hypersecretaory gastropathy has been described[6] in which the histologic changes are similar, except for a larger number of parietal and chief cells than in Menetrier's disease. The gastric acid level is elevated and no protein loss occurs. An intermediate condition has been described in which the clinical and histologic findings are similar to the two previous disorders, but acid production is elevated and protein loss occurs.[7] These three disorders may represent a syndrome with varying clinical and laboratory presenation.

Conclusion

Gastritis is a common gastrointestinal diagnosis. Acute gastritis is treated best by removing the offending agent. Recent studies have shown that acute stress ulcer can be prevented by prophylatic antacids and/or cimetidine. Chronic gastritis is more frequently present, but is rather silent clinically. Pernicious anemia is a frequent concomitant of type A chronic gastritis. Survey for gastric carcinoma should be made if the patient has atrophic gastritis of over ten year's duration.

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AM *Medical Genetics*

Chromosomes and Cancer

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Abstract

Fifteen different chromosomes have been recognized as specific markers of different types of malignancy. These chromosome markers are of importance in the precise diagnosis, therapy, monitoring, and prognosis of cancer.

The idea that cancer may arise from chromosome imbalance was proposed early in this century. Evidence is now abundant that chromosomes and cancer are closely intertwined.

History: The Philadelphia Chromosome

The Philadelphia chromosome occurs in bone marrow cells in chronic myeloid leukemia (CML). The Philadelphia chromosome is a small chromosome, which replaces a normal G (21-22) chromosome. With chromosome banding, it is clear that it is chromosome 22. It is a subtle rearrangement of no. 22. Material from no. 22 is translocated onto another chromosome, usually no. 9

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From 80 to 90% of CML patients have the Philadelphia translocation. The Philadelphia translocation means a better prognosis. CML patients with the Philadelphia chromosome tend to respond to chemotherapy and enjoy longer remissions than other CML patients.

The onset of the acute blast of CML can be predicted by chromosome studies. Additional chromosome markers appear in bone marrow cells as the leukemic cells are transforming for the blast crisis. These signs of impending blast crisis include a change in chromosome 17 and an extra Philadelphia chromosome 22.

Advances in Chromosomes and Cancer

With the advent of chromosome banding techniques, new knowledge has come. Banding involves cooking chromosomes, bathing them in acid or alkali, stretching them, causing them to glow and coloring them with remarkably selective stains. One stain, for example, signals only a part of chromosome 9. Another stain picks out a tiny portion of chromosome 15.

With chromosome banding in 1975 Dr. Barbara Kaiser-McCaw found that changes in chromosome 14 mark a series of lymphomas. Since then the list of chromosomes involved in cancer has grown quickly (Table 1).*

Summary

Chromosomes provide markers for many types of cancer. Chromosome studies are diagnostic, treatment can now be more carefully planned, prognosis more accurately predicted and relapses anticipated and treated. Today knowledge in this field now comprises 15 different chromosomes and a sizeable list of different malignancies in which chromosome studies are useful.

References to chromosome studies of each type of malignancy are available upon request from Dr. Hecht.

Table 1
Chromosomes and Cancer

mphoproliferative Disorders		
Chromosome	Chromosome Change*	Disease
No. 1	Deletion	Lymphoproliferative Disease
No. 5	Deletion	Preleukemia
No. 8	Trisomy	Acute myeloid leukemia
No. 8	Translocation	Lymphomas
No. 9	Translocation	Chronic Myeloid Leukemia
No. 14	Translocation	Lymphomas and Lymphoid Leukemias
No. 15	Translocation	Acute Promyelocytic Leukemia
No. 17	Translocation	Acute Promyelocytic Leukemia
No. 20	Deletion	Preleukemia
No. 21	Trisomy	Acute Leukemia in Down Syndrome
No. 21	Delection	Primary Thrombocytemia
No. 22	Translocation	Chronic Myeloic Leukemia
lid Tumors		
Chromosome	Change*	Disease
No. 3	Translocation	Renal Cell Carcinoma
No. 8	Translocation	Renal Cell Carcinoma
No. 11	Deletion	Wilms Tumor-aniridia
No. 13	Deletion	Retinoblastoma
No. 22	Monosomy	Meningioma
x-related tumors		
Chromosome	Chromosome Change*	Disease
The X	Monosomy	Endometrial Cancer in Estrogen-treated Turner's syndrome
The X	Extra	Breast Cancer in Klinefelter's Syndrome
The Y	Present	Gonadoblastoma in Gonadal Dysgenesis Women with a Y-cell line

eletion = part of chromosome missing. Trisomy = whole extra chromosome. Translocaiton + part of chromosome moved (translocated) onto another chromosome. nosomy = whole chromosome missing.

Neurology

Electroretinography and Oculography

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Electrophysiologic testing of the visual system has received much attention during recent years. Visual evoked potentials (VEP) have become an established means of studying optic nerve function. Perhaps less appreciated is the use of electroretinography (ERG) and electro-oculography (EOG) in assessing retinal function. These studies are complementary, permitting an electrophysiologic assessment of the entire anterior visual system.

Both the ERG and EOG are based upon the presence of a continuous potential difference across the retina such that the cornea is positive with respect to the retina. The ERG records the transient change in this potential induced by a flash of light. The EOG, as done for assessment of retinal function, records the gradual change in this potential proceeding from dark adaptation to light adaptation. Both may be altered by diffuse retinal disease, at times with a pattern characteristic for a specific disease process.

The ERG consists of an initial negative (downward) a-wave generated in the photoreceptor layer followed by a higher amplitude, positive (upward) b-wave that originates in the muller cells (modified glial cells) of the bipolar cell layer (Figure 1). Normal values for amplitude and implicit time must be determined for each laboratory. The implicit time (time from stimulus to wave peak) is measured since the true latency (stimulus to origin of response) is difficult to measure. The ERG is best measured using contact lens electrodes. If contact

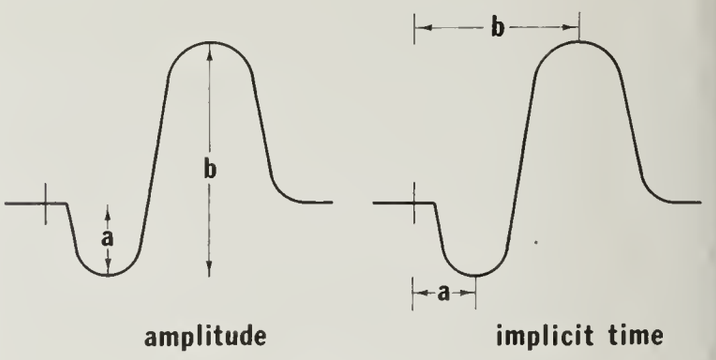


Figure 1
Schematic illustration of the a and b waves of the ERG



Figure 2
Normal ERG, from left to right: light adapted (photopic); dark adapted, weak stimulus (scotopic); dark adapted, strong stimulus; 30 Hz flicker.

lenses cannot be used, a response can generally be obtained recording from a gold covered mylar strip placed in the conjunctival sac. The pupils must be dilated and the corneas anesthetized prior to electrode placement. Infants usually require sedation. The ERG is recorded from each eye following binocular stimulation. Recording made in the light adapted state is termed the photopic ERG (Figure 2). Following about twenty minutes of dark adaptation a low intensity stimulus results in the scotopic ERG. The photopic ERG is predominantly a cone function while the scotopic ERG assesses rod function. A bright stimulus in the dark adapted state results in a mixed rod-cone maximal response and may demonstrate some retinal function when previous reponses were poorly developed or absent. A 30 Hertz flicker is used to test cone function since rods cannot respond at that frequency.

Pure color blindness, a cone dystrophy, is characterized by reduction or absence of the photopic ERG and failure to respond to a 30 Hz flicker. In stationary night blindness, a rod dystrophy, there is a normal photopic but absent or reduced scotopic ERG. Mixed rod-cone dystrophies such as retinitis pigmentosa generally have extinguished or minimal responses under all test conditions, typically before ophthalmoscopically

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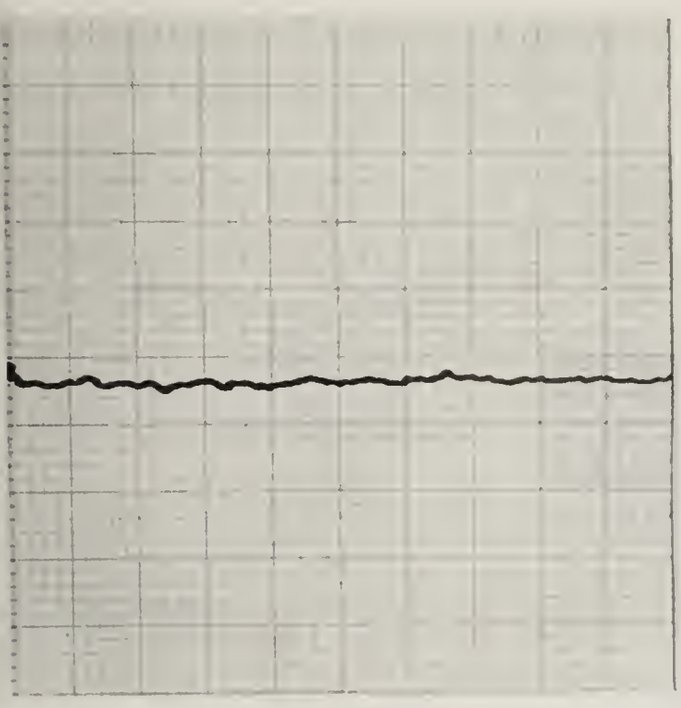


Figure 3
Extinguished ERG from a patient with retinitis pigmentosa.



Figure 4
Selective reduction of b wave amplitude, characteristic of retinal vascular disease.

visible retinal changes are evident (Figure 3). Thus, the ERG is helpful in screening patients with a family history of retinitis pigmentosa. It can also be used in syndromes with pigmentary retinal degeneration such as Usher, Lawrence-Moon-Biedl, Bassen-Kornzweig and Kearne-Sayre syndromes and the mucopolysaccharidoses. Inflammatory pigmentary retinopathies such as that due to rubella or syphilis typically have normal ERG's. Therefore, the test may be used to differentiate acquired pigmentary changes from those of hereditary origin.

In addition to the study of photoreceptor dystrophies, the ERG is increasingly utilized to evaluate vascular, toxic and traumatic retinopathies. Diabetic retinopathy commonly produces an abnormal ERG. It affects the retinal arteries supplying the inner retina and selectively reduces the b-wave while the outer retinal layers supplied by the choroidal vessels are less affected and the a-wave is relatively preserved (Figure 4). This pattern may result from vascular disease of any etiology. ERG changes may be used to follow toxic retinopathies due to chloroquine and Mellaril as well as the toxic effects of intraocular metallic foreign bodies. The ERG is the product of the entire retina and a change in the ERG implicates diffuse retinal disease. Restricted retinal lesions such as maculopathy may cause significant visual loss without altering the ERG. The ERG is normal or supranormal in optic nerve disease.

The ERG may be of considerable diagnostic help in evaluating visual loss in infants and young children. Conditions with characteristic ERG patterns include; 1) Leber's congenital amaurosis (extinguished ERG); 2) ocular albinism (supranormal ERG); 3) total color blindness (abnormal photopic ERG); and 4) congenital stationary night blindness (abnormal scotopic ERG).

The new technique of bright flash ERG has been developed to study retinal function prior to vitrectomy or cataract removal. Opaque ocular media can prevent adequate visualizaton of the retina. The ultrasound examination can help exclude retinal detachment but cannot assess retinal function. The bright flash study accomplishes this. The light from a conventional ERG stimulus may not penetrate the opacity. Therefore, an intense strobe light is utilized with a series of decreasing neutral density filters for graded stimulation. The question of preserved retinal function is especially important in diabetics with vitreous hemorrhage who may or may not have sufficient retinal function to merit proceeding with vitrectomy. Demonstration of a response with the bright flash study may encourage proceeding with surgery.

Electro-oculography is a means of recording eye movement by measuring changes in position of the corneoretinal potential. This is the basis for electronystagmography (ENG) as well as more sophisticated recording for central and peripheral disorders of ocular motility. In addition, the term EOG is used to indicate a test assessing retinal function by measuring changes in the corneoretinal potential. This potential is generated across the retinal pigment epithelium and is altered by disorders of that region. The potential is dynamic and fluctuates with changes in light and dark adaptation. If a light adapted person intermittently looks back and forth between two fixed targets and the room is darkened, there is a slow decrease in the corneoretinal potential and thus in the

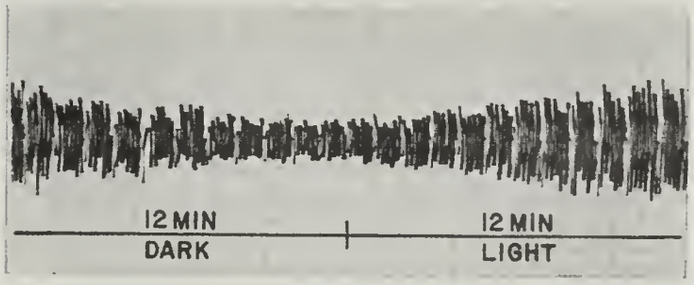


Figure 5
Normal EOG. Amplitude of maximum light adapted response two times that of dark adapted response.

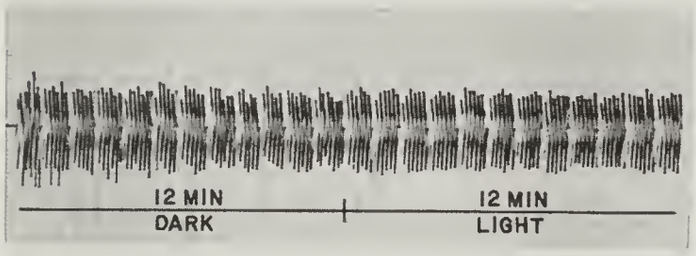


Figure 6
Unresponsive EOG. Patient with Best's dystrophy who had normal ERG.

extent of pen excursion. A minimum is reached in 8-12 minutes. If the room is lighted, the potential then increases to a maximum at 8-12 minutes (Figure 5). The amplitude of the maximum response is normally about twice that of the minimum with the test result reported as the ratio of the two. Values greater than 1.8 are normal. Disease of the retinal pigment epithelium may result in a reduced or completely unresponsive EOG (Figure 6). Many conditions such as retinitis pigmentosa which alter the ERG also affect the EOG. In these instances, the abnormal EOG provides useful confirmation. The EOG may be abnormal while the ERG remains normal. This occurs most often in the flecked retina syndrome and in vitelliruptive macular degeneration (Best's dystrophy). This may also occur in certain toxic retinopathies such as siderosis retinae and chloroquine retinopathy. For these reasons it is generally recommended that both the ERG and EOG be obtained.

Ocular physiology testing has become established as an objective means of assessing visual function. The three dominant tests, ERG, EOG and VEP are all complementary. The ERG and EOG reflect diffuse retinal disease while the VEP best evaluates optic nerve function. The combination of tests permits study of the entire anterior visual system.

Oncology

Radiation Therapy of Early Carcinoma of the Larynx

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In early carcinoma of the larynx, high cure rates ar obtained either with radiation therapy or surgica resection. Radiation therapy preserves the voice, an since most patients with recurrence can still be salvage by surgery, radiation therapy is generally accepted as th treatment of choice for early glottic cancer.

Radiation therapy for this disease requires delivery o a fairly high radiation dose to a small volume of tissue. I order to accomplish this objective, radiation generate by linear accelerators or one of the new generatio Cobalt 60 Teletherapy units is highly desirable. B appropriate fractionation of the radiation dose an protraction of the course over several weeks, most of th acute and delayed radiation reactions can be avoided Subsequent radical surgery can usually be performe safely if the tumor recurs.

The purpose of this article is to present the experience of the Radiation Oncology Division of the Arizona Health Sciences Center (University of Arizona, Tucson, with radiation therapy as the primary mode o treatment for carcinoma of the larynx.

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Table 1
Characteristics of 40 Patients with Early Carcinoma of the Larynx

Characteristic	No. of Patients
Sex	
Male	37
Female	3
Age	
< 50 years	5
> 50 years	35

Table 2
Carcinoma of the Larynx Staging System

T_{1s}	Carcinoma in situ
T_1	Tumor confined to vocal cord(s) with normal mobility
T_2	Supraglottic and/or subglottic extension of tumor with normal or impaired cord mobility
T_3	Tumor confined to the larynx with cord fixation
T_4	Massive tumor with cartilage destruction and/or extension beyond the confines of the larynx

Procedures

During the first 5 years of operation, forty patients with early carcinoma of the vocal cords were referred to the Radiation Oncology Division of the Arizona Health Sciences Center for definitive irradiation. Thirty-seven of the patients were males and three were females, and most of the patients were 50 years of age or older as shown in Table 1. After physical examination and appropriate staging procedures, 29 of the lesions were classified as stage T_1N_0 and 11 as stage T_2N_0. The staging system is described in Table 2.

Most patients were treated with a 4 MeV linear accelerator using 5x5 cm or 6x6 cm fields. The range of total dose is from 6500 to 7000 rads in 6-7 weeks at the rate of 170-200 rads per day. All but one of the patients completed the irradiation course without significant difficulties.

Results

Since 90% of the recurrences and complications appear during the first two years following treatment and the minimum follow-up in our cases is three years, we are confident that our results will not significantly change with extended observation.

Tumor Control:

Three of the 29 patients with T_1N_0 tumors have had disease recurrence (yielding a cure rate of 90%). These three patients underwent laryngectomy and are alive and free of tumor 3-5 years following surgery. Five of the 11 patients (45%) with T_2N_0 lesions recurred locally. Three patients were subsequently salvaged by surgery; the other two died with uncontrolled carcinoma. The total cure rate after radiation therapy and surgery for the radiation failures is 95% as shown in Table 3.

Quality of Voice:

Twenty-five of the 32 patients whose tumor was controlled with irradiation and are available for periodic follow-up evaluations are documented to have excellent speaking voices. Four patients have mild or intermittent hoarseness; however, two of these patients continue to smoke or drink excessively. One patient required a tracheotomy for a complication.

Complications:

Only one of 40 patients (2.5%) developed necrosis of the larynx. Because of faulty technique, he received an excessive dose of radiation—8500 rads—with resultant severe radiation injury to normal tissue. He has now been followed for 40 months after completion of irradiation with a tracheotomy but free of disease.

Discussion

The main objectives of the radiation treatment in cancer of the larynx are: 1) to control the local tumor; 2) to preserve a good quality of voice; and 3) to minimize the incidence of serious complications. Most patients with recurrences can be rescued by surgical resection.

The advantages of a philosophy of treatment in which radiation therapy is the main treatment modality and surgery is used mainly for salvage of radiation failures, are well demonstrated in our study. With radiation it was possible to control the tumor and to preserve phonation in 80% of the patients; the total cure rate after

Table 3
Results

Stage	No. of Patients	Local Control*	No. of Patients (%) Salvaged by Surgery	Total Cure*
T_1N_0	29	26/29 (90%)	3/3	29/29 (100%)
T_2N_0	11	6/11 (55%)	3/5	9/11 (82%)
Total	40	32/40 (80%)	6/8	38/40 (95%)

**3 year minimum follow-up*

salvage surgery in patients with persistent or recurrent disease is 95%.

Normal or near normal voice was reported by 31 of the 32 patients (97%) whose larynx was conserved. Nevertheless, 60% of the patients indicated persistent difficulties such as fatiguing of voice, inability to sing, reduced loudness, hoarseness. Persistence in smoking and heavy drinking during or after the radiation course should be discouraged since it seems to have a detrimental effect on the quality of voice and increases the risk of developing another cancer.

Severe delayed complications like the radiation necrosis of the larynx experienced by one of our patients are infrequent and generally associated with unusually high radiation doses, inadequate fractionation of the radiation dose, and chronic infection.[1] Most patients however will experience mild difficulties during or immediately after the radiation course secondary to mucosal reactions, edema of the arytenoids, or irritation of the adjacent esophagus. The symptoms associated with these reactions—sore throat, hoarseness, dysphagia, dry cough—are usually transient in nature.

Table 4
Second Malignancies

No. of Patients	Tumor Type	Current Status
2	Cancer of the Stomach	Dead
1	Bronchogenic Carcinoma	Dead
1	Colorectal Carcinoma	Dead
1	Nodular Lymphoma	Alive
1	Oral Cavity Cancer	Alive

Individuals with laryngeal cancer are at risk for the development of other neoplasms. Six of our 40 patients have developed second primary tumors and four have died of the second malignancy with no evidence of tumor in the larynx (Table 4). This emphasizes the need for continued follow-up of these patients.

In summary, the use of primary radiation therapy in the treatment of early carcinoma of the larynx and surgery for salvage of radiation failures has cured 95% of the patients and 80% of them have retained vocal function.

Advanced Cancer of the Larynx:

Could a similar approach be applicable to patients with more advanced larynx tumors?

In general, the treatment policy for patients with T_3N_0 and T_4N_0 lesions is primary surgery preceded by or followed by irradiation whenever warranted by clinical, operative, or histological findings. An occasional patient who refuses surgery is referred for primary radiation therapy. Therefore, large series of patients with advanced disease are not available for evaluation of radiotherapy results.

More recently, however, Harwood et al[2] reported the ten-year experience with 107 patients with advanced glottic cancer treated primarily with irradiation at the Princess Margaret Hospital in Toronto. The local control rates were 45% and 56% for stage T_3N_0 and T_4N_0 lesions, respectively. Sixty percent of the radiation failures were salvaged by surgery with low surgical morbidity. The overall tumor control rates were 69% for stage T_3N_0 lesions and 63% for stage T_4N_0 lesions. These figures compare favorably with those obtained by Ogura et al.[3] using laryngectomy: 69% in stage T_3N_0 and 45% in T_4N_0 lesions. However, in the radiation group the voice was preserved in 65% of the patients with T_3N_0 tumors and in 80% of the patients with T_4N_0 tumors.

These results suggest that a philosophy of treatment which emphasizes preservation of the larynx and natural voice is also possible in selected patients with advanced cancer of the larynx without sacrificing survival.

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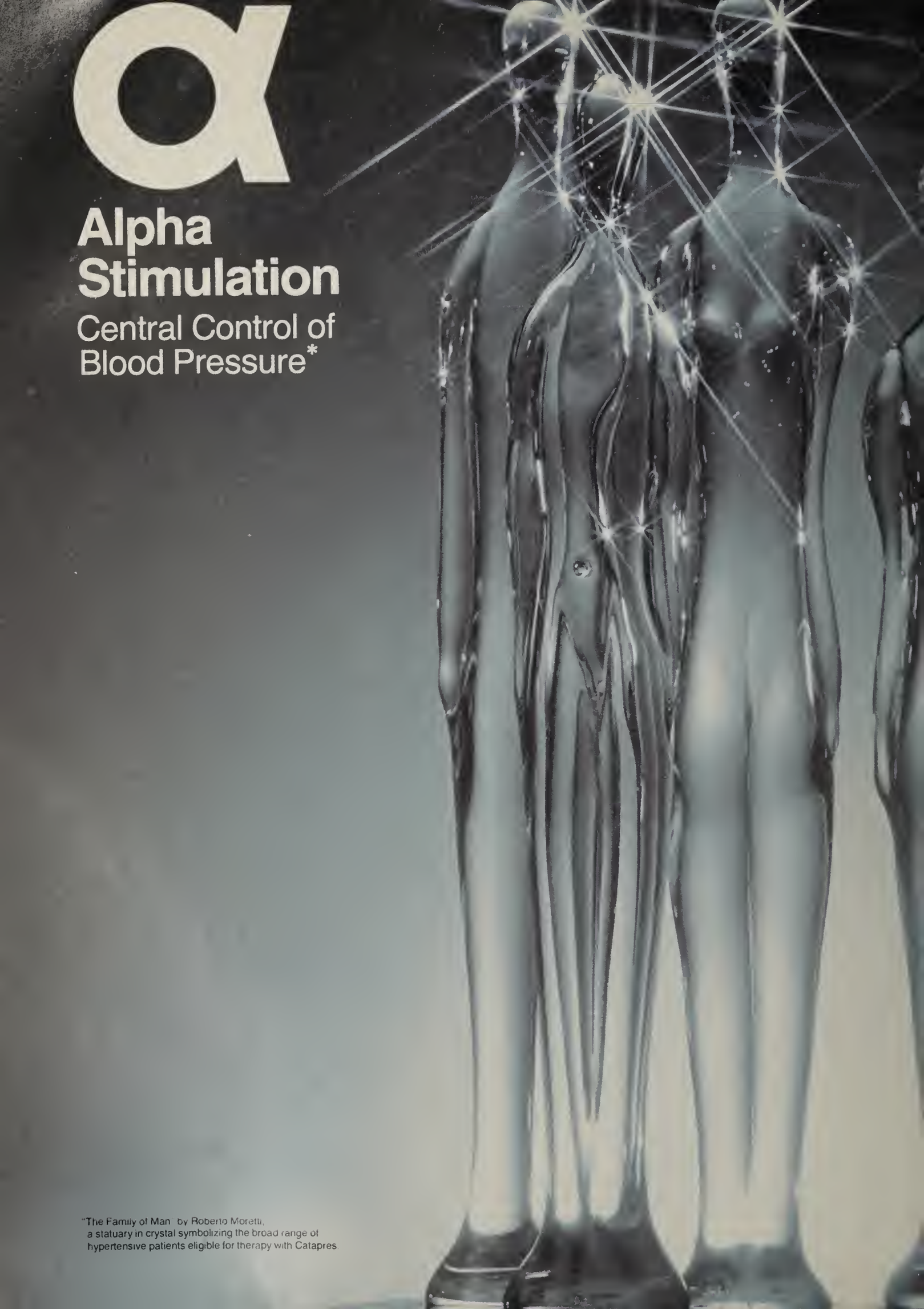
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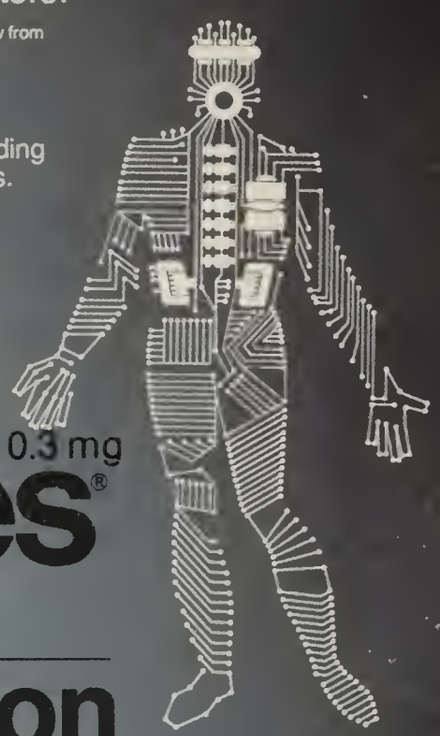
The drug's effectiveness in a given patient, its side effects, warnings, precautions, tolerance, etc. A rational therapeutic choice depends on a careful assessment of all such factors.

*Central alpha-adrenergic stimulation decreases sympathetic outflow from the brain, as shown in animal studies.

[1] Data on file at Boehringer Ingelheim Ltd.

Please see last page for brief summary, including warnings, precautions, and adverse reactions.

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Warnings: Tolerance may develop in some patients necessitating a reevaluation of therapy.
Usage in Pregnancy: In view of embryotoxic findings in animals, and since information on possible adverse effects in pregnant women is limited to uncontrolled clinical data, the drug is not recommended in women who are or may become pregnant unless the potential benefits outweigh the potential risk to mother and fetus.
Usage in Children: No clinical experience is available with the use of Catapres (clonidine hydrochloride) in children.

Precautions: When discontinuing Catapres (clonidine hydrochloride), reduce the dose gradually over 2 to 4 days to avoid a possible rapid rise in blood pressure and associated subjective symptoms such as nervousness, agitation, and headache. Patients should be instructed not to discontinue therapy without consulting their physician. Rare instances of hypertensive encephalopathy and death have been recorded after cessation of clonidine hydrochloride therapy. A causal relationship has not been established in these cases. It has been demonstrated that an excessive rise in blood pressure, should it occur, can be reversed by resumption of clonidine hydrochloride therapy or by intravenous phentolamine. Patients who engage in potentially hazardous activities, such as operating machinery or driving, should be advised of the sedative effect. This drug may enhance the CNS-depressive effects of alcohol, barbiturates and other sedatives. Like any other agent lowering blood pressure, clonidine hydrochloride should be used with caution in patients with severe coronary insufficiency, recent myocardial infarction, cerebrovascular disease or chronic renal failure.

As an integral part of their overall long-term care, patients treated with Catapres (clonidine hydrochloride) should receive periodic eye examinations. While, except for some dryness of the eyes, no drug-related abnormal ophthalmologic findings have been recorded with Catapres (clonidine hydrochloride), in several studies the drug produced a dose-dependent increase in the incidence and severity of spontaneously occurring retinal degeneration in albino rats treated for 6 months or longer.

Adverse Reactions: The most common reactions are dry mouth, drowsiness and sedation. Constipation, dizziness, headache, and fatigue have been reported. Generally these effects tend to diminish with continued therapy. The following reactions have been associated with the drug, some of them rarely. (In so instances an exact causal relationship has not been established.) These include: Anorexia, malaise, nausea, vomiting, parotid pain, mild transient abnormalities liver function tests; one report of possible drug-induced hepatitis without icte and hyperbilirubinemia in a patient receiving clonidine hydrochloride, ch thalidone and papaverine hydrochloride. Weight gain, transient elevation of blo glucose, or serum creatine phosphokinase: congestive heart failure, Raynau phenomenon; vivid dreams or nightmares, insomnia, other behavioral chang nervousness, restlessness, anxiety and mental depression. Also rash, gioneurotic edema, hives, urticaria, thinning of the hair, pruritus not associa with a rash, impotence, urinary retention, increased sensitivity to alcohol, dryne itching or burning of the eyes, dryness of the nasal mucosa, pallor, gynecomas weakly positive Coombs' test, asymptomatic electrocardiographic abnormalit manifested as Wenckebach period or ventricular trigeminy.

Overdosage: Profound hypotension, weakness, somnolence, diminished or sent reflexes and vomiting followed the accidental ingestion of Catapres (clonid hydrochloride) by several children from 19 months to 5 years of age. Gas lavage and administration of an analeptic and vasopressor led to complete covery within 24 hours. Tolazoline in intravenous doses of 10 mg at 30-min intervals usually abolishes all effects of Catapres, (clonidine hydrochloride) ov dosage.

How Supplied: Catapres, brand of clonidine hydrochloride, is available as 0.1 m (tan) and 0.2 mg (orange) oval, single-scored tablets in bottles of 100 and 1000. Al available as 0.3 mg (peach) oval, single-scored tablets in bottles of 100.

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Psychiatric Disorders

Personality Change

William B. McGrath, M.D.

Editor:
Thomas E. Bittker, M.D.

A change in personality is as medically important as an unexplained loss of weight.

By personality change is meant the development of some pattern or trait which is out of character for the individual, or an unhealthy exaggeration of a pre-existing attitude or way of reacting. A few examples:

- An ordinarily inhibited person becomes outgoing, expansive.
- A refined individual shows coarsening of manners or unexpected vulgarity in his use of language.
- A perfectionist becomes careless, procrastinating.
- A heretofore stable person becomes irritable, anxious, suspicious.
- A normally energetic individual becomes listless, uninterested, indecisive.

Any such change may occupy roughly as long as six months. And during this interval any one of a hundred medical or neurological disease may have so far defied clinical or laboratory detection. From 10 to 40% of what appear to be "functional" disorders later prove to have "organic" causation.

Among the conditions which for a while might seem entirely subjective or psychogenic, perhaps the commonest is covert alcohol dependency. The person who cannot relax or sleep without at least a pint of whiskey per day may never appear grossly inebriated or miss time from work. He will minimize to himself and disclaim to you the severity of his dependence. But he will be chronically toxic and he will have vague complaints of malaise, fatigability, dyspepsia, depression and loss of confidence. In time he will be too self-conscious and tremulous to express himself at meetings or make a public appearance.

A related problem is gradually increasing dependency on tranquilizing medicines and proprietary hypnotics, the latter containing bromides or scopolamine.

Neurologic degenerations, endocrine imbalances, primary and metastatic malignancies and a host of systemic diseases often produce changes in personality and subjective complaints for weeks or months before they can be detected or demonstrated objectively. Hungtington's Chorea, brain tumors, disseminated lupus, multiple sclerosis, Alzheimer's disease,—any or all can sometimes and unavoidably be overlooked while psychological investigation is underway.

A relatively high percentage of medical conditions are mistakenly viewed or treated as psychological. This is partly because medical students and residents are not taught to inquire carefully about *changes* in personality. The physician, seeing a new patient or doing an annual physical, has a kind of check-list on a form sheet or in his head. The questions about symptoms are yes-or-no and do not leave room for consideration of changes or gradations.

A more important cause of diagnostic error or delay is the fact that the individual who is undergoing a personality change will often be the last to realize it. Or he will have plausible rationalizations for his personal, social and occupational impairments. The patient

From: 4910 North 44th Street, #A-6

suggests that he has simply been misunderstood or he is somehow a victim of circumstances.

Digression, because here looms a real dilemma. A very brilliant and conscientious physician drove away his patients and ended up with a small, unsuccessful practise. He allowed his patients no little white lies. In trying to obtain as complete and accurate information as possible, he interrogated each individual as if he or she were on the witness stand and as if he or she were somehow morally accountable to him. He made people uncomfortable and they drifted away and never returned.

Predictably he argued that he would not and could not compromise his self-righteous integrity and his judgmental attitudes.

No, there are tactful ways of asking questions and not taking premature issue with repsonses that might not be completely accurate or informative. It is mostly a matter of timing, a delay perhaps until the physician can make it clear that he is not judging but just wanting to understand and to be helpful.

Because the patient undergoing a personality change is likely to minimize or rationalize it, we suggest a feasible technique. The nurse or secretary who fills out the patient information form is instructed to ask the spouse or companion, with sincere professional interest: "Have you noticed any evidences of a personality change?" And she can elaborate, if necessary, about changes in habits or attitudes or moods, commenting diffidently that sometimes a person unwittingly withholds important information from the doctor, and conveying by her manner that everyone involved is just wanting to be helpful.

In the evaluation of a personality change, the psychiatrist can serve a uniquely valuable function. Given a case in which physical and laboratory and x-ray findings are consistently negative, a psychiatric consultant is best qualified to advise whether or not to defer medical judgment in anticipation of the appearance of some pathognomonic sign of disease.

When a psychiatrist reports that a symptom or syndrome does not seem likely to be psychogenic, the referring physician ought surely not take offense. For the psychiatrist will have assessed myriad items and observations which constitute "neurotic loading." He will have asked about alcoholism or suicide or mental illness in the family history. He will be interested in any childhood abuses, deprivations or bereavements. He will infer from the patient's general biography whether there have been evidences of long-standing vulnerability or susceptibility to psychotic illness, as in the personality disorders. In the absence of any clues to such neuropsychiatric vulnerability, the experienced psychiatrist is not the first but the last to presume a "functional" explanation of a personality change.

This is why we recommend that most patients referred to a psychiatrist should be referred for evaluation and not, presumptuously, for treatment.



Case of the Month #48
Left Superior Vena Cava

Jonathan M. Levy, M.D.
Carl J. Impellitier, M.D.
John K. Crowe, M.D.

Editors:
Jonathan Levy, M.D.
Gearld Pond, M.D.

A 56-year old man presented in third degree heart block with a ventricular rate of 40. A transvenous pacemaker was inserted, but adequate pacing could not be obtained. A chest x-ray was taken and is shown (Figure 1.)

What is the course of the pacemaker?

What is the abnormality?

What can be done to assure better pacing?

From: The Departments of Diagnostic Radiology (Drs. Levy and Crowe) and Thoracic Surgery (Dr. Impellitier) Scottsdale Memorial Hospital, 7400 East Osborn Road, Scottsdale, Arizona 85251

Cactus Thorn Injuries to the Extremities:

Their Management and Etiology

Robert R. Karpman, M.D.
Ronald P. Spark, M.D.
Mary Fried, Ph.D.

Editor:
Jack A. Friedland, M.D.

Introduction

The native cacti of Arizona are one of the most beautiful attractions of the Southwest. However, there are some who tend to appreciate them less than others (Figure 1). Although cactus spine injuries are presumably innocuous, several patients present each year to emergency rooms and surgeons offices with more debilitating problems. The exact etiology for this inflammatory response (infection vs. allergic) remains obscure. The purpose of this study was to review the management of patients with severe injury and to explore some of the possible etiologies for the inflammatory responses noted in those patients.

Methods

The first part of the study involved the review of twenty-five patients who presented to either the emergency room or the surgery clinic of the University of Arizona Health Sciences Center following an injury caused by a cactus spine. There were twenty male and five female patients with a mean age of twenty-three years. The mean time of presentation to the facility was eight days with a range from twelve hours to six months. Areas of involvement included the lower extremity in seven patients and the upper extremity in eighteen patients. The most common type of cactus involved was the opuntia or prickly pear followed by the barrel and the cholla. The most frequent presenting signs included non-fluctuant papules which had erythematous bases and were extremely tender on



Figure 1

Continued on Page 853 →

From: Division of Orthopedic Surgery, Department of Surgery, University of Arizona Health Sciences Center, Tucson, AZ 85724, (Dr. Karpman). Department of Pathology, Tucson, Medical Center, Tucson, AZ. (Dr. Spark). Department of Clinical Pathology, Tucson Medical Center (Dr. Fried). Reprint requests to Dr. Karpman.



Figure 1



Figure 2

palpation (Figure 2). There was also marked soft tissue swelling in most involved areas, particularly in the upper extremity. Frequently, there was also a loss of finger motion. In addition, four patients presented with obvious fluctuant masses and were admitted to the hospital for definitive treatment. Diagnostic studies included aspiration of the erythematous areas and culture for bacterial organisms. Radiographs were also obtained of the involved areas, but were of little value. Management of the mild injuries consisted of removal of all obvious spines, primarily with the use of adhesive tape, thorough debridement of the area and application of a compressive dressing as well as oral antibiotics. In those patients who were admitted to the hospital with obvious infection a formal incision and drainage was performed in the operating room accompanied by several days of intravenous antibiotic therapy. The patients were then followed for the next several months until their symptoms had improved.

Results

All of the cultures obtained from the patients with mild injury were negative for bacterial growth. The fluid obtained from the incision and drainage procedures grew primarily staph aureus and enterobacter. Twenty-three of the twenty-five patients treated with the protocol mentioned above had satisfactory results. Results were considered satisfactory if the patient had no subjective or objective disability. The mean recovery period was six days with a range of up to six months. Two patients later returned with retained spines, one with signs and symptoms similar to a Dupuytren's contracture. A repeat exploration was performed and the spines were removed with an uneventful post-operative recovery.

The second part of the study was an attempt to determine the etiology for the inflammatory responses. It consisted of removal of spines in their natural state from a variety of cacti including prickly pear spine, the prickly pear glochid, the barrel cactus, the cholla and the hedgehog. The spines were also taken from various locations on the cactus itself using sterile technique. The spines were then placed on culture plates and cultured for bacteria, acid fast organisms and fungal organisms for ten weeks.

Results

No bacteria or acid fast organisms were found on any of the cultures. Seventeen of the twenty-five cultures, however, grew a fungal organism primarily Alternaria followed by aspirgillis and other miscellaneous fungi. The type of cactus most frequently positive for fungus was the prickly pear spines followed by the prickly pear glochid, the cholla and hedgehog cactus. The location of the spines on the cactus vs. the intensity of growth of the fungus is noted on Table 1. It should be noted that the higher intensities were on the higher part of the cactus as well as the southern orientation.

Discussion

The management of cactus spine injuries in the past has been one based on empiricism. In general, removal of the spines with placement of a compressive dressing as well as prophylactic antibiotics has led to satisfactory results as was found in our study as well. The exact etiology for spine injury, however, remains unclear. The possibilities included infection, an allergic phenomenon or chemical irritation. Although most spines have some type of waxy surface no one has been able to find a source for chemical irritation. An infectious etiology was ruled out by the fact that most cultures are negative following aspiration of involved areas. Lindsay et al.[1] attempted to culture organisms from cacti using animal models, but were unsuccessful. They believe that any organisms that might be present on positive cultures were derived from the patient's own skin prior to perforation. This would explain the four cases that were admitted to the hospital with obvious infections. It should be mentioned that those patients who did have obvious infections either were suffering from chronic diseases such as diabetes or were in highly contaminated areas at the time of the injury. The final alternative and perhaps the most viable is an allergic phenomenon. Schreiber and Shapiro[2] and Whiting and Bristow[3] studied histologic sections obtained from the skin of

Intensity of Growth	Location			
	North	South	High	Low
+3			6	
+2		5	1	
+1	7	4		9

Figure 3

affected patients. Both groups noted the formation of small granulomas usually with the thorn located in the center. It was concluded that an allergic phenomenon had occurred, however, the antigen remained unidentified. This disease has been referred to as Sabra's disease in those who pick prickly pear for a living. The second part of our study may add light onto the antigen which may be causing the allergic reaction. The fungal organism, Alternaria is generally an airborn pathogen which has been increasing in prevalence in dry climates, particularly the Southwest region. It has been known, however, in the past to cause granulomas in the lung similar to those found on the skin biopsy sections in patients who are allergic. Approximately fifty to seventy-five per cent of the population has an allergic response to the Alternaria organism. It seems plausible that the inflammatory response to the cactus spine occurs by the entrance of the Alternaria through the skin followed by an immune response in those patients allergic to the fungus. This would explain why everyone who gets a spine injury does not have a similar response. Further investigations are under way to test those patients with severe injury, specifically for Alternaria.

In summary it was found that the probable source of cactus spine injury is an allergic reaction to the fungal organism Alternaria which is found on the spine in its natural state. The best management for the problem appears to be the removal of the spine and placement in a compressive dressing. It does now appear that antibiotics are helpful in managing this problem unless there is an obvious infection with a fluctuant mass present. Should an infection be present, the obvious treatment is a formal incision and drainage as well as appropriate intravenous antibiotic therapy.

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Special Article

Media-Docs
A Report on the First AMA Health Media Radio/TV Workshop

Ronald P. Spark, M.D.

The uneasiness of endlessly circling O'Hare on route to the AMA Health Reporting Radio/TV Workshop was acutely exacerbated by the discovery that we were sharing hotel space with the American Bar Association. However, I quickly grasped a positive aspect to having such strange bedfellows. It was rather easy to identify the other 65 doctors, dentists, nurses and health reporters attending our workshop as those not wearing three-pieced suits. Ironically, as it turned out, there were a number of legal concerns expressed about "Media-Doc" activity.

My interest in attending was based on the recent formation of a Media Committee in the Pima County Medical

From: Tucson Medical Center, 5301 East Grant Road, Tucson, Arizona. Dr. Spark is chairman of the Media Committee for the Pima County Medical Society, which sponsors "Prescription for Health," a weekly radio interview program.

Society. Our committee had just arranged for a physician-interview type radio show to start this fall. When this first ever AMA media was advertised, I appealed to the Society and the Arizona Medical Association for sponsorship. In justifying this request, our committee felt the course would give us a rapid and effective digest of the "state of the art" and a clearer understanding of our media objectives. Both organizations did lend support because they recognized the absence and need for local physician-directed mass media communication.

The present health media presents a deluge of material often sensational in its overstatement, polarizing and destructive in its effect and, currently, almost immune to effective rebuttal. This health media stance has been fostered by a growing societal background of anit-scientism, anti-elitism and an intense competition to usurp medicine of its professionalism.

In contrast, there are many positive aspects of the meteoric use of the "Media-Doc." In fact one panelist marveled as to why it had actually taken so long to occur. Media surveys rate the highest listener interest in the "fear" and "vanity" categories, i.e., beauty, health and family. While only a few people are sick (and those are already in contact with medicine), most everyone wants health information. Marry the American fascination for science with the perception of the physician as a health expert and your end product results in the highest TV response ratings.

Currently most health reporting is a journalistic beat covered by nonphysicians. Because of the communications training they inherently have trouble distinguishing fairness (equal time for all sides) from veracity (most representative of actuality). Their product is further misproportioned by their managers (editors, producers, etc.) to fit into the time or spatial constraints. The media's distorted emphasis on "maximum impact" often translates into good medicine being perceived as poor copy or bad TV. The public's "right to know" is obviously their primal ethic and takes precedence over any medical, legal or ethical concerns that other professionals might feel compelled to exercise.

In collecting six of the most successful new breed of "Media-Docs (4 MD's and 2 DDS)" for the Workshop, the AMA hoped to encourage physician entry into this venture. Physician-commentators lend both authenticity and veracity, much like the ex-football star sportscaster. The analogy even carries further into the types of reporting activities: play-by-play, color commentary, half-time in depth interviews and coaching clinics on the air.

Barry Kaufman, DMD (NBC-TV Chicago) uses current stories (play-by-play) that tend to take an advocacy posture. His shows tend to focus on a small group of unfortunates. Although he admits this may be intentional overstatement, he feels this focus helps promote correction and tells us how the world "works or doesn't work".

In contrast Art Ulene, M.D. (Today Show) does "soft" programming aimed at health education for large groups (coaching clinics). He avoids the no cure or no useful information topics. G. Timothy Johnson, M.D. (Good Morning America) and Michael Halberstom, M.D. (Cable New Network) fall in between these two extremes, i.e., their formats are more like color commentary or half-time indepth interviews on medical issues.

I came away from the two day sessions with a number of suggestions, regardless of the media vehicle. Plan to cover only a few main points and present them early in your program. Remember time may unexpectedly eliminate the last part of your show. Resist the urge to be the font of all knowledge. Presenting a huge mass of unfocused data is a trap many health reporters fall into. A physician's value is in the perspective he lends to the facts. Don't worry about content skrinkage. If you're clever you'll still be able to use all the material you've collected some other time (for example, write an article on the subject). In cutting back the content, do make sure the shorn product is still coherent and animated. In order to do this surgery, define your content objectives by the perception of your goals, your audience, time constraints and type of program.

What about style and show business? You'll doubtlessly be compared to the TV anchorman or the audience's own physician. Don't worry about this. Most doctors are already good communicators and inherently project an image of concern, confidence and being informed. Your style must be consistent. So select one you are comfortable with, such as being yourself. Be enthusiastic. If you're not, your listeners certainly won't. Reach out to the listener. Remember 30% of medicine is show business: the placebo effect.

As far as topics are concerned, nothing is taboo these days if it is handled appropriately, even humor. Don't accept the producer's caution that the audience level of sophistication is that of 10th graders. My perception is that tenth graders are pretty sophisticated these days and rather outspoken. Don't worry about repetition, especially if it's a good point. Most likely no one was litening the first time you said it anyway.

Bob and Betty Sanders (Chicago News Radio), who have been interviewing doctors and others for years, had some valuable pointers:

1. Use the lay term whenever possible.
2. If the topic dictates be serious in tone, but don't take yourself seriously.
3. Put "how to follow-up" information in your presentation.
4. Make sure you follow-up on every unanswered question (and have the resources to do this).
5. Remember advice is always easy to give when you're not directly involved. Therefore, never say never; always say it is possible that . . .; and (the classic) call him back if it gets worse.

Dr. Ulene shared his own operational rules with us. He said the most important thing to remember is that the station owns the antenna. They are not in the health business; they sell time. Although they need public service shows to hold license, they won't allow good medicine if it's bad TV. Lastly, he cautioned, don't stop working or you'll lose credibility with the station and your peers.

Several panelists spent time on the problem of peer acceptance. It is often said that the only way to get a physician to accept anything new is to package it as a bad land deal. Peer envy and prejudice against the positions you propound will only ameliorate slowly. Your goal must be the gained confidence of the physician community. The time of disaffection can be abbreviated by seeking frequent and varied counsel, especially from detractors. Try to convince them it is better to have you on the air than some charlatan.

Legal concern over increased liability exposure was addressed by Neil Chayet, J.D. (CBS "Looking at the Law"). He counseled that your contract must assign full fiscal responsibility for liability protection to the station. (On the other hand no one was aware of their malpractice premiums being hiked because of their activities). The contract must give you complete ownership of your material and allow for reuse of the tapes for any educational purposes. Your tapes will also be a record of what was said—for that future day in court. (It was of interest that none of the panelists had such a contract). Chayet strongly advised the placement of a disclaimer into the program content ("your own physician may have a different view point"). Be careful and responsible in what you say (indiscretion seems to run rampant when physicians enter corridors or step before microphones). If you must use your own cases, disguise it; but not to a degree that you're being dishonest.

The Media Workshop was a success from many standpoints. Those already "in the business" felt their instincts were largely correct in what they were already doing. Most of the 40 or so uninitiated, including myself, left feeling ready to take the plunge.

Radiology Continued

Answer: Persistent Left Superior Vena Cava.

Left sided superior vena cava is one of the most frequent anomalies of systemic venous return, occurring in approximately 0.5% of the population. Most often, embryologic failure of development of the left innominate vein causes persistence of the left anterior cardinal vein, which becomes the left superior vena cava (LSVC.) This vessel almost always drains into the coronary sinus, which enters the right atrium (Figure 2.) Therefore, no venous admixture results.

In most patients, LSVC is of no clinical significance, although it may be associated with other cardiac anomalies. The vessel may produce widening of the superior mediastinum, which may be mistaken for a mediastinal mass. Most often, this widening is subtle and undetected on chest x-ray. The anomaly usually becomes evident with medical intervention, such as with central venous pressure monitoring, right heart catheterization or pacemaker insertion as in our patient. LSVC may cause significant problems during cardiac bypass, especially if its existence is not known prior to surgery.

Once a persistent LSVC is identified, the site of venous access should be directed to the right upper extremity. The great majority of patients with LSVC also have a right superior vena cava, and this vessel drains normally into the right atrium. The right side can therefore be used for correct placement of CVP catheters and pacemaker electrodes. This was done in our patient, and adequate pacing was established.



Figure 2

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State of the Art

Adult Salicylate Intoxication

Kenneth A. Conrad, M.D.

Editor:
Eric H. Prosnitz, M.D.

Introduction

Intoxication with salicylates continues to be a significant problem among adults in Arizona. In 1980, 495 episodes of salicylate poisoning were reported to the Arizona Poison Control Center. One-half of these occurred in adults, and one adult death has occurred. It is gratifying to know that the introduction of safety caps on aspirin bottles has significantly reduced the incidence of poisoning in children. Unfortunately, this measure has not altered the incidence of severity of poisoning in adults, many of whom ingest salicylates intentionally.[1]

The magnitude of the problem of salicylate intoxication in adults is further illustrated by the experience of the University of Colorado Medical Center.[2] Seventy-three adults with salicylate poisoning were admitted to either the Colorado General Hospital or the Denver General Hospital in a 30 month period. Six deaths attributable to salicylate intoxication occurred in this group of patients. Among these 73 patients, two groups were identified: patients (n=53) in whom the diagnosis was apparent upon admission, and the remaining patients (n=20) in whom the diagnosis was delayed. The first group was younger and consisted entirely of intentional overdoses. The second group was older; only 20% of these patients had ingested salicylates in an intentional overdose. The remaining patients in the second group had developed intoxication as a result of salicylate use for associated medical problems. Five of the six deaths which occurred in this series were in the patients in whom diagnosis was delayed.

Pathophysiology

In order to treat victims of salicylate intoxication effectively, the practitioner must appreciate the pathophysiology of the disorder. In adults, acute ingestion of 150 mg/kg of salicylate may produce significant toxicity and/or death. In a 70 kg adult, this amount of salicylate (10 grams) is contained in 30 5 grain aspirin tablets or in 8 cc of methyl salicylate - oil of wintergreen. If many tablets of aspirin are ingested acutely, absorption of the drug may be delayed for many hours.[3] Salicylate initially causes gastrointestinal symptoms - nausea, vomiting and abdominal pain. Although some drug may be eliminated by vomiting, patients who ingest a sufficient quantity of the drug will still develop other systemic signs and symptoms, particularly disturbances of acid-base balance. If salicylate is ingested alone, respiratory alkalosis usually develops first, as a result of stimulation of the respiratory center. Ingestion of other drugs, particularly sedatives or other nervous system depressants, reduces the frequency of respiratory alkalosis and increases the frequency of respiratory acidosis. However, metabolic acidosis (either alone or in combination with respiratory alkalosis) develops eventually in most poisoned patients.[4] Acidosis which is accompanied by an increased anion gap (a difference between the serum sodium and the sum of the serum bicarbonate and chloride of more than 18 meq) results from several factors, including the acid nature of the drug itself, the formation of organic acids such as lactate, and the reduction of excretion of the acids due to depression of renal function. Metabolic acidosis from salicylates must be differentiated from that due to other disorders (Table 1.) Dehydration and sodium loss occur as a result of vomiting, fever and hyperpnea. Potassium depletion occurs as a consequence of respiratory alkalosis and chloride depletion due to

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Table 1
Causes of Metabolic Acidosis
Associated with an Increased Anion Gap

1. Renal Failure
2. Ketoacidosis
3. Lactic acidosis
4. Poisoning
 a. Salicylate
 b. Methanol
 c. Ethylene glycol
 d. Paraldehyde

vomiting. Disorders of blood coagulation occur as a result of alteration in platelet function and inhibition of synthesis of the vitamin K dependent clotting factors. The prothrombin time will become prolonged over a period of hours or days. Salicylate produces central nervous system dysfunction including restlessness ("salicylate jag"), hallucinations, seizures and coma. Tinnitus occurs in many patients with high salicylate levels, but is not a reliable indicator of toxicity since some patients with high levels will not develop tinnitus. Finally, noncardiac pulmonary edema, unrelated to fluid therapy, may occur.[1]

Patients who take aspirin chronically may develop toxicity insidiously, without ingesting large quantities of the drug at any one time.[2] The capacity of the body to form salicyluric acid and the phenolic glucoronide becomes saturated at low doses of salicylate; and the elimination half-life of the drug increases as the dose is increased. During chronic high-dose therapy with salicylate, most of the drug is excreted unchanged in the urine. Small increases in the dose may result in disordinate increases in plasma concentration and in the likelihood of toxicity.

Diagnosis

To detect salicylate poisoning, the physician must maintain a high index of suspicion for this problem. A patient who claims to have ingested salicylate or who is found next to an empty bottle of aspirin tablets is no diagnostic problem. However, the patient with no history of salicylate ingestion who presents with vomiting, confusion, or metabolic acidosis may not be suspected of having salicylate intoxication. Treatment may be delayed, increasing the seriousness of the intoxication.

Salicylate exposure is likely if the ferric chloride test is positive: A few drops of ferric chloride added to 5 ml of urine acidified with HCl will produce a red-violet color if salicylate is present. Ketones will produce a positive test, but boiling the urine will eliminate this false-positive. A positive urine dipstick test for ketones also provides a clue to the possibility of salicylate exposure. Confirmation of the diagnosis is readily established in most major hospital laboratories by requesting a serum salicylate level. Because of the frequency of ingestion of several drugs, it is suggested that toxicologic screening studies be done.

Treatment

Treatment of the intoxicated patient is designed to prevent or reverse the systemic toxicity described in the preceding section. In a patient in whom acute ingestion is suspected, prevention of absorption of salicylate should be attempted. This is best accomplished by induction of emesis by Ipecac (if the patient is conscious and has an adequate gag reflex) or by gastric lavage with a large bore (Ewald) tube. The stomach should be emptied even if the patient claims to have vomited at home. The time elapsed since ingestion is not important in the decision to empty the stomach. After gastric emptying has been accomplished, activated charcoal in a dose of at least 50 grams, should be given by mouth or by nasogastric tube. Charcoal binds salicylate effectively and will prevent absorption of the drug. Serum electrolytes and arterial blood gases should be measured to determine the acid-base status of the patient, and an initial salicylate level should be measured. Although charcoal should prevent absorption of salicylate, the salicylate level should be measured again 2-4 hours later to be sure that there is no absorption of any drug which may be preceding the charcoal in the small intestine.

Supportive management of salicylate intoxicated patients depends primarily upon correction of dehydration and acid-base and electrolyte disturbances. Systemic alkalinization benefits the patient in two ways: salicylate is shifted from the tissues to the blood and salicylate is trapped in the urine if the urine is alkalinized by administration of systemic alkali. Shifting salicylate out of the central nervous system is helpful in reducing central nervous system toxicity which is a major factor in the morbidity and mortality from salicylate.[5] Remember that by inducing systemic alkalosis, plasma salicylate levels will *increase* temporarily. However, this is a desired effect because it is the tissue salicylate (especially that in the central nervous system) which is most deleterious.

As has been mentioned before, the capacity of the liver to metabolize salicylate is saturated at relatively low doses of the drug. Therefore, elimination of the drug depends upon renal excretion. If the serum salicylate level exceeds 400 mcg/ml on admission, therapy aimed at enhancing salicylate elimination is recommended. Renal excretion is enhanced by increasing urine flow and by maintaining the urine pH at 7 or 8. By maintaining a high urine pH, salicylate is kept in its ionized form which is resistant to reabsorption by the renal tubules.[6] Establishing and maintaining an alkaline diuresis is accomplished by first replacing deficits of sodium, potassium and fluid which are often present upon admission to the hospital or emergency room. Thereafter, administration of 500 ml/hr of 5% dextrose in water to which 2 ampules (100 meQ) of sodium bicarbonate have been added will rapidly increase urine flow and urine pH. Potassium may be added to the intravenous fluid in amounts appropriate to the clinical situation. Acetazolamide should *not* be used to alkalinize the urine because it may cause systemic acidosis and

actually increases morbidity in animals with salicylate poisoning.[5] Patients with normal renal function and with no history of congestive heart failure should tolerate this amount of fluid and sodium without difficulty. Careful monitoring of serum electrolytes, urine flow and pH, serum salicylate and arterial blood gases will assure that a diuresis has been achieved and that salicylate levels are falling.

Treatment of the patient with chronic salicylate intoxication may require all of the measures described above, particularly if the patient is confused or has metabolic acidosis. On the other hand, if the patient has only elevated salicylate levels without clinically significant toxicity, discontinuation of salicylate may be sufficient therapy.

If the patient has significant underlying renal dysfunction or congestive heart failure, and alkaline diuresis is deemed to be inappropriate, salicylate elimination may be enhanced by hemodialysis or hemoperfusion.[7,8] Hemodialysis has the advantage that it also enables the practitioner to correct electrolyte disturbances quickly. Most adults will improve clinically with the supportive measures described. If the patient's clinical status deteriorates despite these measures, extracorporeal removal of salicylate may be necessary.

Summary

The diagnosis of salicylate intoxication can be made easily if the physician suspects it. In particular, elderly patients who present to the physician confused, with volume depletion and/or metabolic acidosis should be suspected of being intoxicated with salicylate. Deaths from this poisoning can be prevented if management is begun promptly and if close attention is paid to correction of fluid and electrolyte imbalance. Most patients with acute salicylate intoxication will respond to gastric emptying and administration of activated charcoal, combined when appropriate, with alkaline diuresis. Patients who have chronic salicylism will require discontinuation of salicylate and depending upon the clinical situation, treatment aimed at reducing salicylate levels more promptly. Hemodialysis and hemoperfusion should be restricted to the minority of patients who cannot tolerate alkaline diuresis or who fail to respond to it.

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Editorials

Beware of a New Form of Hypercalcemia

Hypercalcemia has become a more prevalent condition with the institution of automated chemistry screening. As such, many more patients are now being evaluated for this disorder in the hopes of finding a treatable cause. Of the many conditions known to cause hypercalcemia, the most difficult to diagnose is still primary hyperparathyroidism due to autonomous secretion of parathormone. To this day the most reasonable way to evaluate such a patient is to exclude all other causes of hypercalcemia, and then invoke primary hyperparathyroidism as the etiology of the patient's problem. If this is not difficult enough, it has now become apparent that there is another form of hypercalcemia which mimics that of primary hyperparathyroidism in which parathyroid gland exploration is not indicated. It is this newly recognized disorder that needs greater attention.

In *familial hypercalciuric hypercalcemia*, chronic hypercalcemia generally begins before the age of ten years, and is not accompanied by hypercalciuria or renal damage. It is an autosomal dominant disorder with near 100% penetrance. It is thought by some to be a benign disorder in which the usual secondary effects of hypercalcemia do not become apparent, i.e., development of renal stones, nephroclacinosis, peptic ulcer disease and pancreatitis. None of the patients so far reported have had other endocrine tumors which are known to be associated with primary hyperparathyroidism in the multiple endocrine adenoma syndromes.

The underlying etiology of the condition is not clear at this time. Levels of parathyroid hormone are not usually elevated, and levels of urinary cyclic AMP do not distinguish patients with this disorder from those with primary hyperparathyroidism. In most cases, hypercalcemia persists following parathyroid gland excision and histologically the glands removed show hyperplasia. The exact prevalence of this disorder in the general population is unknow since many patients are incorrectly classified as having primary hyperparathyroidism. Perhaps, this disorder accounts for the patients who continue to have hypercalcemia following parathyroidectomy when no discrete parathyroid adenoma is found at the time of surgery.

In order to correctly classify patients who have hypercalcemia, in whom other causes of hypercalcemia have already been excluded, measurement of urinary calcium excretion is essential. It has been recommended that a simple diagnostic index of renal calcium clearance to creatinine clearance should be obtained with a value below 0.01 suggesting familial hypcalciuric hypercalcemia. Hypocalciuria in such a patient should lead to a study of other family members to see if they demonstrate similar findings. Since surgical intervention in such situations has not proven beneficial, these patients should be followed conservatively inasmuch as their morbidity tends to be quite low. Hopefully, with greater sophistication in our understanding of calcium metabolism, we will develope better diagnostic indices for this condition.

For the time being, urinary calcium excretion measurements over several days seems a worthwhile endeavor to distinguish those patients with familial hypocalciuric hypercalcemia from those thought to have primary hyperparathyroidism.

Marshall B. Block, M.D.
Editor

Geriatrics at the University of Arizona

Population projections for the United States indicate a major increase in the number of persons reaching age 65 during the next twenty years. This group currently represents some 11% of our population and is expected to reach 16-18% by the turn of the century. Arizona is a retirement state and 14% of the residents in its urban areas are already over age 65. The Governor's Task Force on Retirement has predicted that by the year 2000, one out of every four Arizonans may be over the age of 65.

This rapidly enlarging group uses health resources to a greater extent than other adults. They commonly have multiple medical problems, many of which are chronic; they spend more time in the hospital; and they consume a major amount of health practitioner time. Persons in this later stage of life often have social, environmental, and emotional problems which complicate the course of medical illness. Twenty-eight percent of all personal health care expenditures in 1977 was for the over 65 individual and 67% of public funds for health care was utilized by this group. Half of the patients cared for by primary care physicians are over the age of 65 and in many specialties this proportion is much higher; i.e., urology, orthopedics, psychiatry, and cardiology.

In the past, many medical schools have not adequately prepared their students to deal with the special set of circumstances surrounding their elderly patients. Many study groups, particularly the National Academy of Sciences Institute of Medicine, have recommended the inclusion of geriatrics in the curriculum for all medical students and in the training programs for all physicians. The Institute's report recommended the development of a cadre of faculty to teach gerontology and geriatrics and an increase in appropriate content on aging in basic and clincial science courses. It further recommended that preparation for care of the aged be included in the clinical clerkships and in housestaff training programs, as well as in examinations for certification and licensure. The report also urged expansion of aging research in the bio-behavioral sciences, clinical medicine, and health services research.

In response to these needs, the University of Arizona has embarked on a broad course designed to implement those recommendations. Its Committee on Gerontology has a major responsibility to coordinate and guide educational and research efforts and has been highly successful in stimulating new research activities in gerontology and geriatrics with the full support of the University administration. The Department of Internal Medicine has created a Section of Geriatrics and similar changes are occurring in the Departments of Family and Community Medicine and Psychiatry. Faculty positions specifically for the development of curriculum and clinical training programs in geriatrics and gerontology have been identified in each of these three departments. The Curriculum Committee of the College of Medicine is conducting a survey of aging content in basic and clinical science courses. Gaps in this important area of the curriculum will thus be identified and plans for enhancement can be made. Elective clerkships in geriatrics during the senior year have been implemented in the Departments of Internal Medicine, Family and Community Medicine, and Psychiatry. Rotation through a geriatric assessment and planning ward is now a

required part of the Internal Medicine Residency Training Program. The Univeristy of Arizona Colleges of Medicine and Business and Public Administration have been successful in their application for an Administration on Aging (AOA) Title IV Planning Grant leading to the development of a geriatric center. Only twenty-two of these planning grants were funded throughout the United States. They were designed to improve clinical services for the frail elderly with chronic illness, improve geriatric training, and stimulate research activities. An expanded grant request was recently submitted to fund a progression from the planning phase to the operational stage of a geriatric center for the State of Arizona.

Health care for older individuals must be seen as interdisciplinary. The Arizona Health Sciences Center is making every effort to coordinate its activities in geriatrics and gerontology with other colleges in the University. In particular, the Colleges of Nursing and Pharmacy have active programs in geriatric education and training and will represent an important part of this interdisciplinary approach to long-term care for the elderly. Emphasizing the concern for the social and health needs of the frail elderly the University is supported in development of the geriatric center concept and in implementing its fundemental goals by a wide interest and great expertise in the local community and in the state. It is hoped that over a period of time both the education component of the health professions relative to the elderly and the provision of comprehensive and sophisticated care for the elderly will be improved. Graduates with a positive attitude towards the needs of the elderly and highly developed knowledge and skills in the provision of these services is the ultimate goal of the University of Arizona. Equally important is understanding the complex needs of the elderly patient and acquisition of knowledge to assemble and coordinate the resources, both medical and social, to aid our senior citizens to maintain function, to prevent deterioration, and to provide them with compassionate and skillful long-term care.

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Editor—

The article "Snake Venom Poisoning, Diagnosis and Treatment" by Thomas W. Boyden (*Arizona Medicine*, Vol. 37, No. 9, pg 639) fails to make a point of great importance. Whereas antivenin treats the systemic symptoms of rattlesnake envenomation rather well, it treats the local tissue destructive effects of the proteolytic enzymes poorly. I have seen a number of patients who have been treated "properly" with antivenin who nonetheless have suffered considerable tissue destruction at the site of the bite. When this occurs on the hand, morbidity is considerable. Often multistage reconstructive efforts to provide skin coverage, tendon grafting or nerve grafting are required.

The use of excisional therapy in the management of snake bite has been well outlined by Huang et al in the *Annals of Surgery*, May, 1974, pages 598-607. Immediate surgical excision of the envenomated tissue, sparing vital structures removes the envenomated area and avoids the use of antivenin. This type of therapy is mistakenly identified as "fasciotomy" by Dr. Boyden. A careful reading of the article "Early Debridement in Pit Viper Bite" by Thomas G. Glass, Jr., *Surgery, Gynecology and Obstetrics*, May, 1973, pages 774-776, and the numerous other publications by Dr. Glass shows that he is **not** advocating fasciotomy as treatment of snakebites. He is advocating prompt excision of the envenomated tissue and fasciotomy only as specifically indicated.

There is unquestionably a place for antivenin therapy. The patient showing systemic signs of envenomation, and the patient with signs of severe local envenomation are cadidates for antivenin therapy in addition to other measures. The majority of other patients can b managed without antivenin.

Joshua J. Tofield, M.D.
Tucson, Arizona

Editor—

The letter by Dr. Tofield refers to patients with rattlesnake envenomation, "properly" treated with antivenin, subsequently having "considerable tissue destruction at the bite site." Without knowing the details of his cases I cannot comment on whether or not "proper" antivenin treatment failed to antagonize the local effects of injected venom. Important variables contribute to the severity and outcome of any envenomation. These include the "first-aid" given to the victim (application of tourniquets or ice, physical activity of the victim following the bite, incision and/or suction of the bite site), the amount of antivenin given and the time elapsed between the bite and the administration of antivenin. As stated in my article the majority of experienced physicians would emphasize the efficacy of antivenin in neutralizing the local tissue effects of injected rattlesnake venom.

Despite the title of the most recent article by Dr. Thomas Glass, ("Early Debridement of Pit Viper Bites" *JAMA* 235:2513, 1976,) a careful reading of the methods section indicates that the fascia at the bite site is routinely opened and the "fasciotomy" is either terminated or extended after inspection of the tissues. In the series of 54 patients reported by Huang et al, nine had postoperative surgical complications including necrosis of skin grafts, osteomyelitis, amputation and joint contracture. **All** of the patients treated in the fashion advocated by Dr. Glass or Huang et al have tissue destruction and permanent scarring. Some patients will have severe loss of limb function. The fact that these authors use a surgical approach to the patient with snake venom poisoning does not constitute proof of efficacy when compared to no specific therapy or antivenin administration.

Dr. Tofield and I are in agreement that patients with systemic signs of envenomation and patients with signs of progressive local envenomation need antivenin. Fortunately, the majority of patients with snake venom poisoning need no specific therapy. I remain opposed to the routine use of any surgical treatment (other than for incision and suction) in cases of snake venom poisoning.

Thomas W. Boyden, M.D.
Tucson, Arizona

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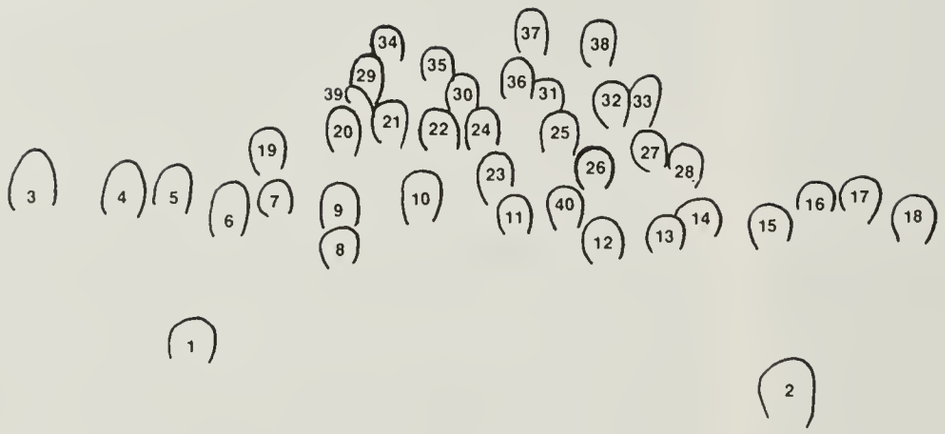
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In 1952 the 9982 ORTs TSU General Hospital of Phoenix, Arizona comprised of physicians and nurses and ancillary personnel from Arizona spent two weeks active duty training at fort Huachuca, Arizona.

This group's photograph of the officers and nurses assumes some historical value thirty years later.

See next page for names→



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10. Maj. Joseph C. Constantine, MSC, Tucson
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12. Lt. Col. Hyman Fischer, MC, Tucson
13. Maj. Philip Greenbaum, MC, Tucson
14. Lt. Col. Solomon Natzer, MC, Tucson
15. Unidentified
16. Lt. Col. Leon L. Titche, MC, Tucson
17. Lt. Col. Frank G. Drischel, MC, Tucson
18. Maj. Charles A. Janda, MC, Tucson
19. Capt. Ralph L. Hoag, MSC, Tucson
20. Capt. Bernard L. Rosenbaum, MSC, Tucson
21. 1st. Lt. Joseph R. Powell, Jr., MSC, Phoenix
22. WOJG. Leonard E. Eschelbach, Phoenix
23. Unidentified
24. Capt. Edmundo F. Felix, MC, Tucson
25. Col. Justin E. Gains, MC, Tucson
26. Lt. Col. Samuel R. Kates, MC, Phoenix
27. Capt. Wallace H. Branaman, MSC, Tucson
28. Col. Donald A. Polson, MC, Phoenix
 Commanding Officer of the Unit
29. Unidentified
30. Col. Seymour Fischer, MC, Phoenix
31. Lt. Col. John W. Kennedy, MC, Phoenix
32. Maj. Wesley G. Biddulph, DC, Phoenix
33. Lt. Col Kenneth G. Rew, MC, Phoenix
34. WOJG. Ralph L. Sage, Phoenix
35. 1st. Lt. Jess L. Thompson MSC, Phoenix
36. Maj. Abe Adlund, MC, Phoenix
37. Lt. Col. Ruland Hussong, MC, Phoenix
38. Maj. Weymouth Pew, MSC, Mesa
39. Capt. Mary Ellen Carter, ANC, Phoenix
40. Unidentified

Not Shown:

Maj. Ernest A. Borne, MC, Prescott
Col. Robert M. Stump, MC, Phoenix

ArMA Reports

The minutes appearing in this section have been edited to conserve space. A complete copy of the minutes of any meeting will be mailed to any member requesting them.

AD HOC LONG RANGE PLANNING COMMITTEE

The meeting of the Ad Hoc Long Range Planning Committee of the Association held at 810 West Bethany Home Road, Phoenix, Arizona , on Monday, September 29, 1980, convened at 7:04 p.m., John E. Oakley, M.D., Chairman, presiding.

Charge of Committee

The committee, being charged with developing goals for the Association which could be accomplished in a specified period of time, and developing programs whereby those goals could be achieved, discussed at great length the existing primary objectives set fourth in the Articles of Incorporation as well as the current problems facing the Association and its members at the present time.

In light of the many priorities which seem to exist and which could certainly be addressed by the committee in its selection of goals, it was determined that each committee member submit, in writing, a list of objectives or purposes felt by each to be of ultimate importance. All responses would be assimilated for the committee's review and consideration during its next meeting.

Additionally, it was suggested that the goals and guidelines set by other associations be solicited for the committee's review, in conjunction with those of its members, to allow a broader overview of avenues available for future planning.

Next Meeting

The next meeting of the committee will be held in November, subsequent to the meeting of the House of Delegates scheduled for November 1, 1980, since the outcome of that meeting would be a definite factor in the committee's deliberations.

EXECUTIVE COMMITTEE

A meeting of the Executive Committee of the Arizona Medical Association, Inc., held in Room 2216 of the Basic Sciences Building at the U of A College of Medicine in Tucson, Arizona, on Saturday, October 4, 1980, a quorum being present, convened at 8:14 a.m., John T. Clymer, M.D., President and Chairman, presiding.

BOMEX Rule Making Process

Rule on Excessive Fees

The committee reviewed BOMEX memorandums dated September 23 and September 26, 1980, together with a letter from M. David Ben-Asher, M.D., dated September 26, 1980, and discussed at great length the establishment of a rule on excessive fees. Along this line, counsel agreed to provide the members with a summary of background facts and information on this matter for the committee's review prior to final hearings on the rule which would be held at some future date.

Other Rules

It was brougt to the committee's attention that BOMEX is in the process of reviewing all of its rules and it was determined that a review of these rules should be made to determine the Association's position, if any, on specific sections thereof, so that should recommendation by ArMA be indicated, it might be made prior to final action by BOMEX. It was the consensus of the members that the matter be referred to the Professional Committee for review and comment.

Review of Board of Directors' Agenda

The agenda for the Board of Directors' meeting to be held October 4, 1980, was discussed, item by item, with additions and changes being made.

Other Business

Proposition 106

Pamphlets and brochures containing informative material on Proposition 106. *Received.*

Statewide Health Coordinating Council

The committee reviewed the request of the Bureau of Health Planning, Arizona Department of Health Services, for nominations for membership on the Statewide Health Coordinating Council (SHCC) and, following discussion,

It was moved and carried that the Arizona Medical Asssociation submit the name of Louis C. Kossuth, M.D., to the Bureau of Health Planning as its nominee for membership on the Statewide Health Coordinating Council (SHCC).

Definition of Death

Counsel's memorandum relating to Statutory Definition of Death dated October 1, 1980. *Received.*

BOARD OF DIRECTORS

A meeting of the Board of Directors of the Arizona Medical Association, Inc., held in the Student Lounge of the Basic Sciences Building at the U of A College of Medicine in Tucson, Arizona, on Saturday, October 4, 1980, a quorum being present, convened at 10:06 a.m., John T. Clymer, M.D., President and Chairman, presiding.

Welcoming Remarks

Louis J. Kettel, M.D., Dean of the College of Medicine at the University of Arizona, welcomed the members of the Board and their guests, expressing his delight at being able to host the day-long schedule of activities and commenting briefly on the newest developments occurring at the College.

Schedule of Events

Schedule of events for the balance of the day's activities. *Received.*

Lowell H. Steen, M.D., Chairman of the Board of Trustees of the American Medical Association, arriving at the meeting following press conferences, was introduced to the Board as the speaker for the Official Family Briefing session to be held later in the day.

Auxiliary Activities Report

Mrs. Joyce Newman, President of the ArMA Auxiliary, briefly reported to the Board on the successful Fall Leadership Conference the Auxilliary had recently held in Sedona, during which workshops were held relating to HSAs, speaker training, graphics and cost containment. The brochures, "Is He Sick? Shall I Send Him to School?" having now been printed in Spanish, as well as English, were being distrubuted to the schools throughout the state. The Board members were furnished copies of each and, should they desire, could obtain additional copies for their office waiting rooms. Additionally, the Auxiliary was hard at work with the "Early Start to Good Health" program, preparation of a handbook, planning for annual meeting and various other projects.

ArMPAC Activities Report

In the absence of Dr. Dale, Dr. Clymer reported that ArMPAC had recently distributed a total of $9,000.00 to various state legislative campaigns.

Board of Directors

United States Pharmacopeial Convention

Consent of Gerald C. Moczynski, M.D., to serve as ArMA's representative on the United States Pharmacopeial Convention. *Received.*

Boddicker vs. Arizona State Dental Association

Court decision and advice of appeal in Boddicker vs. Arizona State Dental Association. *Received.*

Certifice of Need

The Board reviewed the proposed Joint ArMA/ArHA Position Paper on Certificate of Need, as well as received comments from Coconino, Cochise, Graham, Greenlee, Maricopa and Pima County Medical Societies relative thereto. Brian Lockwood, Chairman of the Board of ArHA, addressed the members regarding the background for the drafting of the proposed paper and, following lengthy discussion.

It was moved and carried that the proposed Joint ArMA/ArHA Position Paper on Certificate of Need be referred back to the committee initially drafting same for further study and review.

ExecutiveCommittee

Ratification of Minutes

It was moved and carried to approve and ratify the minutes of the meetings of the Executive Commitee held on August 4, 1980, August 8, 1980, and September 19, 1980.

Membership Classification Changes Approved

1. Paul S. Causey, M.D. - Active to Active Over 70 - Account Age - Effective 10/4/80 - Dues Exempt.
2. Norman A. Ross, M.D. - Associate to Fifty-Year Club.

American Medical Association

Task Force "Future Directions for Medical Education"

Acknowledgement of ArMA's recommendation of Rudolf Kirschner, M.D., for appointment to the AMA Task Force "Future Directions for Medical Education." *Received*

AMA Councils

The AMA's request for nominations for membership on AMA Councils was reviewed and

It was moved and carried that, subject to his consent, Edward Sattenspiel, M.D., be submitted as ArMA's nomination for membership on the Council on Legislation.

It was determined that this matter should be brought back as an agenda item for the January 10, 1981, meeting of the Board, should any of the members wish to submit additional recommendations for any of the councils named.

Research & Education Committee

Activities reported by Earl J. Baker, M.D., on the steps being taken by the Research & Education Committee to inform the Delegates and Alternate Delegates on all aspects of unified membership prior to the special meeting of the House of Delegates of ArMA scheduled for November 1, 1980. *Received.*

Benevolent & Loan Fund Committee

Letter of appreciation from Gerardo Carlos. *Received.*

Finance Committee

Financial Statement

Financial Statement for period ending 8/31/80. *Received.*

1981 Committee Budgets

The Board reviewed the recommendations from the Finance Committee relating to the 1981 Committee Budgets and, following discussion,

It was moved and carried to approved the 1981 Committee Budgets as follows:

500A	Articles of Incorporation and Bylaws	$ 500.00
500B	Benevolent & Loan	6,000.00
500D	Board of Directors	4,200.00
500E	Executive	3,000.00
500F	Finance	500.00
500H	Grievance	-0-
500I	History & Obituaries	-0-
500J	Maternal & Child Health	1,000.00
500K	Health Manpower	800.00
500L	Legislative	18,000.00
500M	Medical Education	3,000.00
500N	Medical Economics	1,000.00
500O	Occupational Health	-0-
500Q	Professional	3,500.00
500R	Public Relations	20,000.00
500S	Governmental Services	2,000.00
500U	Housestaff	1,000.00
500W	ArMA Auxiliary	1,000.00
500X	Physician Rehabilitation	3,500.00
Total 1981 Committee Department Budget		$ 69,000.00

During the review of committee budgets, much discussion centered around the Benevolent & Loan Fund Committee budget and the House of Delegates' action taken on May 1, 1964, following which the Board unanimously reaffirmed the Association's previous position relating to interest income, *i.e.*, all accumulated interest shall be credited to the Benevolent & Loan Fund Committee for use in the awarding of scholarships.

Accounting Services - Arizona Perinatal Trust

The Board reviewed and approved the Finance Committee's recommendation that all future accounting services provided by ArMA to the Arizona Perinatal Trust would be provided at a cost to the Trust.

Annual Meeting Registration Fees

After consideration of the September 27th action of the Finance Committee relating to a request from the Scientific Assembly Committee for consideration of a dues increase to allow elimination of member registration fees for annual meetings,

It was moved and carried to approve the recommendation of the Finance Committee to reject the recommendation of the Scientific Assembly Committee for a review of ArMA's present dues structure and consideration of a dues increase which would include a sufficient amount to allow elimination of ArMA meeting registration fees.

Maternal & Child Health Care Committee

Arizona Perinatal Trust

Declaration of Trust of the Arizona Perinatal Trust dated August 29, 1980. *Received.*

Board of Trustess - APT

Pursuant to the recommendation of the Executive Committee made in meeting on September 19, 1980,

It was moved and carried to designate William C. Scott, M.D., as a member of the Board of Trustees of the Arizona Perinatal Trust.

Professional Committee

Confirmation of Appointment

It was moved and carried to confirm the appointment of Thomas P. Foerster, M.D., as Chairman of the Professional Committee's section on Sports Medicine.

Statutory Definition of Death

The Board received the Professional Committee's earlier Resolution 3-80 relating to Statutory Definition of Death, together with informational materials and a memorandum of legal counsel dealing with this issue; and, following a review of all documents,

It was moved and carried to refer back to the Professional Committee, for further study and review, all of its documentation dealing with the Statutory Definition of Death, together with a memorandum of opinion of legal counsel on this issue dated October 1, 1980.

Other Business

Southern District Director

The Board received the resignation of Joseph S. Whaley, M.D., as Southern District Director, effective 12/31/80 and Pima County's recommendation that Clifford D. DeBenedetti, M.D., be appointed to such position until the 1981

In Hypertension*...When You Need to Conserve K^+

Every Step of the Way



Each capsule contains 50 mg. of Dyrenium® (brand of triamterene) and 25 mg. of hydrochlorothiazide.

†Step 1 usually consists of an initial phase (a diuretic alone), a titration phase (dosage adjustment and/or addition of a K^+ supplement or K^+-sparing agent), and a maintenance phase (a diuretic alone or in combination with a K^+ supplement or K^+-sparing agent).

Serum K^+ and BUN should be checked periodically (see Warnings).

Before prescribing, see complete prescribing information in SK&F Co. literature or *PDR*. A brief summary follows:

* **WARNING**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded; in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in postsympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia, although uncommon, has been reported. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. Dyazide' should be withdrawn before conducting tests for parathyroid function.

Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

Adverse Reactions: Muscle cramps, weakness, dizziness, headache, dry mouth, anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances. Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and, rarely, allergic pneumonitis have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components.

Supplied: Bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.

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NEOSPORIN® Ointment

(polymyxin B-bacitracin-neomycin)

Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

works just as well in their homes.

- It's effective therapy for abrasions, lacerations, open wounds, primary pyodermas, secondarily infected dermatoses.
- It provides broad-spectrum overlapping antibacterial effectiveness against common susceptible pathogens, including staph and strep.



- It helps prevent topical infections, and treats those that have already started.
- It contains three antibiotics that are rarely used systemically.
- It is convenient to recommend without a prescription.

NEOSPORIN® Ointment—for the office, for the home.
(polymyxin B-bacitracin-neomycin)

Effective • Economical • Convenient • Recommendable

Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Annual Meeting of the House of Delegates.

It was moved and carried to accept the resignation of Joseph S. Whaley, M.D., as Southern District Director, effective December 31, 1980, and to appoint Clifford D. DeBenedetti, M.D., to serve as such Southern District Director from January 1, 1981, until elections are held during the Annual Meeting of the House of Delegates in May, 1981.

Special Mention

This being the first meeting of the Board subsequent to the AMA Annual Meeting at which Dr. Cloud was able to be present, the members of the Board offered unanimous congratulations to him upon his success in seeking the office of President-Elect of the American Medical Association.

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Chapter, American Cancer Society
Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Arizona Thoracic Society/Arizona Lung Association
Walter O. Boswell Memorial Hospital Sun City
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Memorial Hospital of Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, (THMEP) Tucson
University of Arizona College of Medicine, Tucson
Veterans Administration Medical Center, Phoenix
Veterans Administration Hospital, Prescott
Yuma Regional Medical Center

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

1981
JANUARY

Eight Annual Symposium
Barrow Neurological Institute of St. Joseph's Hospital, & Medical Center - Controversies In Neurology and Neurosurgery
January 8-10, 1981. La Posada Resort, Scottsdale, Arizona. Sponsor: St. Joseph's Hosptial and Medical Center, and the Barrow Neurological Foundation. Contact: Richard A. Thompson, M.D., Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013. Approved for 15 hours category 1 credit.

Respiratory Allergy Immunology
January 9, 1981, Walter O. Boswell Memorial Hospital, Sun City, Sponsor and Contact: Martha R. Brennan, Ed.D., Dir. of Education, Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for 1 hour of category 1 credit.

Postgrad Course In the Diagnosis & Treatment of Headache
January 9-11, 1981. Mariott's Camelback Inn, Scottsdale, AZ. Sponsor: American Assn. for the Study of the Headache. Contact: Am. Assn. for the Study of Headache, 5252 N. Western Ave., Chicago, Ill. 60625. Approved for 15 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
January 13-15, 1981, Arizona Medical Association Offices, Phoenix, Arizona. Sponsor: ACEP, Arizona Affiliate - Am. Heart Assn., & ArMA. Contact: Carol Tuner, ArMA 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for hour per hour category 1 credit

Personal Instruction Course in Catract Surgery
January 14-17 and 28-31. The Foundation, Pheonix, Arizona. Sponsor and Contact: The Eye Foundation, 421 N. 18th Street, Suite 105, Phoenix, AZ 85006. Approved for hour per hour category 1 credit.

Ninth Annual Surgical Symposium
January 19-23, 1981. Del Webb's La Posada Resort Hotel, Scottsdale, AZ. Sponsor: Maricopa County General Hospital and Phoenix, Surgical Society. Contact: Phoenix Surgical Society, 333 W. Thomas Rd., Suite 209, Phoenix, AZ 85013. Approved for 31 hours of category 1 credit

Clinical Recognition and Management of Heart Disease 1981
Twenty-Fourth Annual Scientific Session of the American Heart Association, Arizona Affiliate
January 21-24, 1981. University of Arizona College of Medicine, Main Auditorium, Tucson, AZ. Sponsor: University of Arizona College of Medicine, American College of Physicians and the Arizona Heart Association. Contact: Office of CME and Outreach U. of A. Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Workshop Series in Family Therapy
January 23 & 24, 1981, Scottsdale Camelback Hospital, Scottsdale. Sponsor: Philadelphia Child Guidance Center and Arizona Family Learning & Communication Center. Contact: Arizona Family Learning & Community Center, Metro Medical Plaza, B403, 3201 W. Peoria Ave., Phoenix, AZ 85029.

Common Problems with the Hand & Upper Extremity
January 27, 1981. Walter O. Boswell Memorial Hospital, Sun City AZ. Sponsor and Contact: Dept. of Education, Walter O. Boswell Memorial Hospial, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for hour per hour category 1 credit.

Advanced Cardiac Life Support Program (Recertification)
January 28, 1981. Arizona Medical Association, Phoenix, Arizona. Sponsor: ACEP, Arizona Affiliate - Am Heart Assn., & ArMA, Contact: Carol Turner, Arizona Medical Association, 810 W. Bethany Home Rd., Phoenix, AZ 85013.

A.A.F.P. Annual Scientific Meeting
January 29-31, 1981. Doubletree Inn Mall, Scottsdale, AZ. Sponsor: Arizona Academy of Family Physicians. Contact: Lee Mahoney, 8534 E. Amelia, Scottsdale, Arizona 85251. Approved for 18 hours of category 1 credit.

Southwest Desert Symposium In Anesthesiology
January 30-Feb. 1, 1981. Marriott Hotel, Tucson, AZ., Sponsor: U of A College of Medicine Tucson. Contact: Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for category 1 credit.

FEBRUARY

7th Annual Meeting
Freestanding Ambulatory Surgical Association
February 5-7, 1981. Doubletree Inn, Scottsdale, AZ. Sponsor: Arizona Medical Assn. & the Society of Freestanding Ambulatory Surgical Association. Contact: Brian Dawson, M.D., 1040 E. McDowell Road, Phoenix, AZ 85006. Approved for 16 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
February 10 & 11, 1981. Arizona Medical Association, Phoenix. Sponsor: ACEP, Arizona Affiliate - Am. Heart Assn., & ArMA. Contact: Carol Turner, Arizona Medical Association, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Rural Emergency Medicine
"The First 60 Minutes"
February 12 & 13, 1981. Doubletree Inn, Scottsdale, AZ. Sponor: Arizona Chapter American College of Emergency Physicians, AZ Affiliate - Am. Heart Assn. & ArMA. Contact: Carol Turner, ACEP, Executive Secretary, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 13½ hours of category 1 credit.

Seventh Annual Frontiers in Ophthalmology
February 12-14, 1981. The Arizona Biltmore Resort. Sponsor: St. Luke's Hospital Medical Center. Contact: Christine Campbell, Seminar Coordinator, St. Luke's Hospital. 525 N. 18th St., Phoenix, Arizona (602) 258-7373, Ext. 311. Approved for 15 hours of category 1 credit.

Physical Diagnosis Skills
February 14, 1981. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for 6 hours of category 1 credit.

Third Annual Practical Counseling Technique Workshop for the Primary Physician
February 14-21, 1981. Lion Square Lodge, Vail, Colorado. Sponsors: Institute for Human Relationship and Development and Colorado Academy of Family Physicians. Contact: John C. Faul, M.D., 5290 E. Yale Circle, Ste 200, Denver, Co. 80222.

Third Annual International Catarct Surgery Symposium
February 16-18, 1981. La Posada Resort Hotel, Scottsdale, Arizona. Sponsor: The Eye Foundation (A.F.O.R.E.) Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th Street, Ste 105, Phoenix, AZ 85006. Approved for 12 to 22 hours of category 1 credit.

International Cardiovascular Congress III
February 16-18, 1981. Scottsdale Center for the Arts, Scottsdale. Sponsor: Arizona Heart Institute. Contact: Internation Cardiovascular Congress III, Arizona Heart Institute, P.O. Box 10,000, Phoenix, AZ 85064. Approve for 18 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider)
February 18-20, 1981. Arizona Medical Association. Sponsor: ACEP, Arizona Affiliate - Am. Heart Assn., & ArMA. Contact: Carol Turner, Arizona Medical Association, 810 West Bethany Home Rd., Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Endocrinology 1981
February 19 & 20, 1981. Arizona Biltmore Hotel, Phoenix. Sponsor: St Joseph's Hospital and Medical Center and the Flinn Foundation. Contact: Daniel S. Duick, M.D., Dept. of IM, St. Jospeph's Hospital and Medical Center, P.O. Box 2071. Phoenix, AZ 85001.

Emergency Care Update-1981
February 19-21, 1981. U of A Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, U of A Health Sciences Center, Tucson, AZ 85724. Approved for hour per hour category 1 credit.

Ninth Annual Taos Lung Disease Symposium
February 20-22, 1981. Holiday Inn, Taos, New Mexico. Sponsor: New Mexico Chapter of the American Thoracic Society. Contact: New Mexico Chapter of the American Thoracic Society, 216 Truman, NE, Albuquerque, New Mexico 87108.

EKG Interpretation and Arrhythmia Management
February 20-21, 1981. Hyatt Regency Hotel, Phoenix, AZ. Sponsor: International Medical Education Corp. Contact: Stephen Mattingly, Vice President, International Medical Education Corp., 64 Inverness Dr. East, Englewood, Co. 80112. Approved for 13 hours of category 1 credit.

Fourth Annual Mid-Winter Symposium in OB/GYN
February 25-27, 1981. Scottsdale Hilton Hotel, Scottsdale, Arizona. Sponsor: Maricopa County General Hospital, Dept. of OB/GYN, and Phoenix OB/GYN Society. Contact: Secretary, Phoenix OB/GYN Society, 333 E. Virginia, Ste 222, Phoenix, AZ 85004. (602) 257-9218.

Personal Instruction Course in Cataract Surgery
February 25-28, 1981. The Eye Foundation, Phoenix, AZ. Sponsor: The Eye Foundation. Contact: The Eye Foundation, 421 N. 18th Street, Suite 105, Phoenix, AZ. Approved for hour per hour category 1 credit.

Prophlactic Antibiotics in Surgery
February 27, 1981. Boswell Memorial Hospital, Sun City, AZ. Sponsor and Contact: Office of CME, Walter O. Boswell Memorial Hospital, 10401 Thunderbird , Sun City, AZ. Approved for hour per hour category 1 credit.

14th Annual Southwestern Clinical Pharmacy
Clinical Aspects of Psychotherpauetics
February 27-March 1, 1981. Ramada Inn, Tucson, AZ. Sponsor: U of A College of Pharmacy. Contact: Jack R. Arndt, Ph.D., College of Pharmacy, U of A, Tuson, AZ 85721. Approved for 8 hours of category 1 credit.

MARCH

5th Annual Valle of the Sun Dermatologic Symposium
March 4-9, 1981. Scottsdale Hilton, Scottsdale. Sponsor: American Academy of Dermatology. Contact: Phoenix, Dermatologic Society, Jeffrey M. Grant, M.D., 13000 N. 103rd. Ave., Ste 50, Sun City, AZ 85351. Approved for 12 hours of category 1 credit.

Crisis and Emergency Pediatrics
March 5-8, 1981. Doubletree Inn, Tucson. Sponsor: U of A College of Medicine. Contact: Hugh C. Thompson, M.D., Pediatrics, U of A College of Medicine, Tuson, Az 85724. Approved for 17 hours of category 1 credit.

Gynecologic Endocrinology
March 6-8, 1981. The Adams Hotel, Phoenix. Sponsor: U of A College of Medicine and Center for Continuing Professional Education. Contact: David Pent, M.D., P.O. Box 33772, Phoenix, AZ 85067. Approved for 14 hours of category 1 credit.

Advanced Cardiac Life Support Progran (Provider)
March 10-12, 1981. Arizona Medica Association. Sponsor: ACEP, Arizona Affiliate - Am. Heart Assn., & ArMA. Contact Carol Turner, Arizona Medical Association 810 West Bethany Home Road, Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Crisis and Emergency Pediatrics
March 5-8, 1981. Doubletree Inn, Tucson, AZ Sponsor: U of A College of Medicine, and Department of Pediatrics. Contact: Hugh C Thompson, M.D., Pediatrics, U of A Health Sciences Center, Tucson, AZ 85724 Approved for 17 hours of Category 1 credit

Annual Meeting—Society of Head and Neck Surgeons
The American Society For Head and Neck Surgery
March 8-11, 1981. The Hyatt Regency Hotel, Phoenix, Arizona. Sponsor: American Society of Head and Neck Surgery. Contact: Gabriel F. de Freitas, M.D., Locall Arrangements Chairman, Suite 101, 1901 E. Thomas Road, Phoenix, AZ 85016.

Personal Instruction Course in Catract Surgery
March 11-14 and 25-28. The Eye Foundation, Phoenix, Az. Sponsor and Contact: The Eye Foundation, 421 n. 18th Street, Suite 105, Phoenix, AZ 85006. Approved for hour per hour category 1 credit.

Chronic Obstructive Pulmonary Disease
March 12, 1981. Walter O. Boswell Memorial Hospital, Sun City. Sponsor and Contact: Walter O. Boswell Memorial Hospital, 10401 Thunderbird Blvd., Sun City, AZ. 85372. Approved for 1 hours of category 1 credit.

Seventh Annual Maricopa County General Hospital
Urological Seminar
March 14 & 15, 1981. The Registry Resort, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: James H. McDonald, M.D., Chief of Urology Service, MCGH, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 14 hours of category 1 credit.

Third Annual Symposium on Current Concepts in the Managment of Acute and Chronic Pain
Mrach 14 & 15, 1981. Camelback Inn, Phoenix. Sponsor: Maricopa County General Hospital. Contact: George Wallace, M.D., Program Director. MCGH, 2601 E. Roosevelt, Phoenix, AZ 85006.

3rd Annual Conference on Adjuvant Therapy of Cancer
March 18-21, 1981. Tucson Convention Center, Tucson. Sponsor: U of A College of Medicine. Contact: Cancer Center Division, Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 25 hours of category 1 credit.

Ski CME Program
March 21-26, 1981. Vail, Colorado. Sponsor: Desert Samaritan Hospital. Contact: L.A. Rosati, M.D., CME Committee, Dester Samaritan Hospital, 1400 S. Dobson Rd., Mesa, AZ. Approved for 16 hours of category 1 credit.

Advanced Cardiacl Life Support Program (Recertificatlon)
March 26, 1981. Arizona Medical Association, Phoenix. Sponsor: ACEP, Arizona Affiliate - Am. Heart Assn., & ArMA. Contat: Carol Tuner, Arizona Medical Association, 810 West Bethany Home Rd., Phoenix, AZ 85013. Approved for hour per hour category 1 credit.

Prophylactic Antibodies in Surgery
March 27, 1981. Walter O. Boswell Memorial Hospital, Sun City. Sponsor and Contact: Walter O. Boswell Memorial Hospital, Office of CME, 10401 Thunderbird Blvd., Sun City, AZ 85372. Approved for 1 hour of category credit.

APRIL

Vectorcardiography (An Advanced Workshop)
April 29, 30 & May 1, 1981. Sponsor: American College of Cardiology and Good Samaritan Hospital Institute for Cardiovascular Diseases. Contact: Registration Secretary, Extramural Programs Department, American College of Cardiology, 9111 Old Georgetown Rd., Bethesda, MD 20014.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Ravi Koopot, M.D.

Clinical Conference Cardiovascular Medicine
Third Tues.,5:15 p.m., second floor classroom

ARIZONA STATE HOSPITAL

2500 E Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed.. 1.00-2:00 P.M., J-6 Conf. Rm., Contact. Dr Conger & Staff

Clinical-Pathological Conference
3rd Wed., 1.30-2:30 P.M. General Services Bldg., Conf. Rm.

Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgical Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third and 5th, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists

Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.

Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

WALTER O. BOSWELL MEMORIAL HOSPITAL

10401 Tunderbird Boulevard, Sun City, AZ 85372. Contact: Martha R. Newby, ED.D., Director of Education.

Medical Department Continuing Medical Education
4th Wednesday, 12 Noon, C119. May, July, Sept. & Nov.

Tumor Board
Last Thursday of the month, 12 Noon, Educ Center Classroom I & II, May, July, Sept, & Nov.

Surgical Department CME
4th Friday, 7 A.M., Educ. Center Classrooms I & II. Contact: Brian Updegraff, M.D.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor Camelback Hospital. Contact: T. F. Reid, Jr., M.D.

Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A Rosati, M.D. Approved for category 1 credit.

CME Programs
Weekly Tuesday, 7 p.m., Cafeteria-West End.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 p.m. Buffet Lunch, Grande Rm.

OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 p.m. Grande Rm.

Pediatric Case Conference
2nd. Friday, 12:30 p.m., Grande 2

DOCTORS HOSPITAL

1747 East Thomas Road, Phoenix, Arizona 86016 Contact: Medical Staff Secretary for additional information.

Physicians Continuing Education Program
1st Thursday, 12:30 p.m., Classrooms.

EL DORADO HOSPITAL TUCSON (THMEP)

1400 N. Wilmont Road, Tucson, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: W. J. Howard, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 p.m., S-4 Conf Rm.

Gynecological Section Conference
2nd Monday, 12:30-1:30 p.m., West Rm

Monday Noon Conference
Weekly, 12:00-1:00 p.m. R-2 Conf. Rm.

Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 p.m., West Rm.

FRONTIERS IN OPHTHALMOLOGY 1981

The seventh annual **Frontiers in Ophthalmology** seminar will explore the latest research and techniques in significant areas of ophthalmologic care. A distinguished faculty has been selected to conduct the seminar sessions.

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This seminar meets the criteria for 15 hours of C.M.E. credit.

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For additional information, write or call:
Christine Campbell, Seminar Coordinator/St. Luke's Hospital Medical Center
525 N. 18th Street, Phoenix, Arizona 85006/(602) 258-7373 ext. 311

Obstetrical Section Conference
1st Monday, 12:30-1:30 p.m., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 p.m., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 p.m., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:30-8:30 a.m., Contact: Paul Bergeson, M.D.
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 a.m. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 p.m., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 p.m., D-5 North.
Dr. Fenger's Lecture
Weekly, Wed., 12:30-1:30 p.m., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 a.m., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 p.m., OBGYN Conf. Rm.
PM & R Grand Rounds
2st. & 4rd. Wed., 12:00-1:00 p.m., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 p.m., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 p.m., F151, Radiology Conf. Rm.
Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 a.m., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 p.m., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 p.m., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 p.m., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 a.m., West Rm.
Neuropathy Conference
Last Friday, 1:15-2:15 p.m., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 p.m. , East Rm.
PM & R Journal Club
Alt. Fridays, 12:00-1:00 p.m., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL,(THMEP)

2800 E. Ajo Way, Tucson, AZ. 85713. Contact: Eric C. Ramsay, M.D., Approved for Category 1 credit.
Attending's Hour
5th Monday, 12:30 p.m., Contact: J. Goldberger, M.D.
Infectious Disease Conference
3rd Monday, 12:30 p.m., Contact: Barry Friedman
Ophthalmology Conference
1st Monday, 12:30 p.m., Contact: Bruce Gillers, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: J. Santiago, M.D.
Surgical Connference
Weekly Mondays, Contact: R. Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 p.m., Contact: B. Phibbs, M.D.
Clinical Pharmacology Conference
5th Tuesday, 12:30 p.m., Contact: M. Katz, Clinical Pharm.
Neurology Conference
1st & 3rd. Tuesday, 12:30 p.m., Contact: J. Laguna, M.D.
Radiology Conference
2nd Tuesday, 12:30 p.m., Contact: M. Baker, M.D.
Medical Surgical Grand Rounds
2nd Wed., 12:30 p.m., Contact: Chief Medical Resident
Medical Conference
1st Wed, 12:30 p.m., Contact: Chief Medical Resident
Cardiology Conference
1st Thursday, 12:30 p.m., Contact: B. Phibbs, M.D.
Endocrine Conference
2nd Thursday, 12:30 p.m., Contact: E. Munjack, M.D.
GI Conference
4th Thursday, 12:30 p.m., Contact: C. Kanun, M.D.
Mortality & Morbidity Conference
3rd. Thursday, 12:30 p.m., Contact: E. Munjack, M.D.
OB/GYN Dept. Meeting
2nd Thursday, 2 p.m., Contact J. Wurzel, M.D.
OB/GYN Pathology Conference
Weekly,Thursday, except 2nd, Contact: J Wurzel, M.D.

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MUTUAL INSURANCE COMPANY OF ARIZONA Sponsored by the Arizona Medical Association.

OB/GYN Sonograph/X-ray Conference
4th Thursday, 3 p.m., Contact: J. Wurzel, M.D.
Clinical Pathology
5th Thursday, 12:30 p.m., Contact: J. Fleishman, M.D.
Renal Conference
1st Friday, 12:30 p.m., Contact: T. Pence, M.D.
Hematology Conference
2nd & 4th Friday, Contact: S. Ketchel, M.D.
Rheumatology Conference
3rd Friday, 12:30 p.m., Contact: J. Goldberger, M.D.
Chest Conference
5th Friday, 12:30 p.m., Contact: D. Engelsberg, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008
Continuing Medical Education Program
Every other Wednesday, 7-8 a.m.

Clinical Conferences
2nd & 4th Wednesday, 12-1 p.m., Medical Staff Lounge

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, AZ 85008. Contact: Leonard Tamsky, M.D.
Medicine Conference
Daily 12-1 p.m., Contact: S. Schaffner, M.D.
Chest Surgery Conference
Weekly, Mondays, 1:30 p.m., Santa Cruz Rm.
Hematology Conference
2nd Tuesday, 1:00 p.m., Contact: Walt Koppenbrink, M.D.
OB/GYN Dept. Grand Rounds
Weekly Tuesday, 12 Noon, Santa Cruz Room
Obstetrical Problem Conference
Weekly Tuesday, 7:30 a.m., Yavapai Rm.
Orthopedic Conference
Weekly Tuesday, 7:30 a.m., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 a.m.,Contact: Robert Ganelin, M.D.
Urology Discharge Planning Conference
Weekly, 11:30 a.m., Station 42
Hand Surgery Conference
Weekly Wed, a.m., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 p.m., Station 42.
OB/Neonatology Seminar
Weekly, Wed. 7:30 a.m., Yavapai Rm.
Clinical Psychiatric Conference
Weekly, Wed., 11-12 p.m., Mental Health Annex, Rm. 1346.
Surgery Conference
Weekly Wed., 7-8 a.m., Surgical Dept.
Current Concepts in Medicine & Surgery
1st Thursday, 1 p.m., Dr. Hospital Class Rm., Contact: Dr. Tamsky.
Cardiology Conference
Weekly Thurs., 2 p.m., Santa Cruz Rm.
OB/GYN Resident Conference
Weekly Thurs., 12 p.m., Yavapai Rm.
GYN Endocrine Seminar
1st & 3rd Friday, 12:30 p.m., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:30 a.m., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 a.m., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MEDICAL CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact; James L. Grobe, M.D., Approved for category 1 credit.
Clinical Conferences
1st & 3rd Tuesdays, 12 noon, 5th floor auditorium.
CPC or Medical-Surgical Forum
4th Tuesday, 12 noon, 5th floor auditorium.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St., Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D., Approved for category 1 credit.
Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 a.m., Contact: D. Randall, M.D.
Clinical Staff Teaching Conference, Rm. A.
Wed. Weekly, 8:00 a.m.
Otolaryngology Grand Rounds
4th Wednesday, 4-5 p.m., Conference Rm. A, Contact: N. Wendell Tood, M.D.
Family Practice/Emergency Room Teaching Conference
Thursday Weekly, 7:30-8:30 p.m., Conf. Rm. A, Contact: Drs. L. Fairbanks & E.Y. Hooper.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for category 1 credit.
Monthly Medical Education Seminar
3rd Monday, 6:30 p.m., Kiva Conf. Rm.
Clinical Conferences
Weekly Tuesday, 12:30 p m., Kiva Conference Rm.
Tumor Board Conference
Weekly Friday, 12 p.m., Kiva Conf. Rm. Contact: H. Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th Street, Scottsdale, AZ 85251. Contact: W. S. Williams, M.D., Approved for category 1 credit.
Family Practice Conference
1st Monday, 12:30 p.m., Doctors' Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 p.m., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 p.m., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:30 p.m., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 p.m., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 p.m., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 p.m., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 p.m., Doctors' Lounge
Cardiolpulmonary Conference
4th Tuesday, 12:30 p.m., Doctors' Lounge
OB/GYN Dept. Meeting & Conf.
2nd Wed., 12:30 p.m., Doctors' Lounge
Trauma Conference
3rd Thurs, 12:30 p.m., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 p.m., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 p.m., Doctors' Lounge
GI Medical—Surgical-Radiological Conf.
2nd Friday, 12:30 p.m., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 p.m., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.
OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 p.m., 1st Floor Conf. Rm.
Genetics Conference
Weekly Monday, 12:30 p.m., Pediatric Department.
Pediatric Rounds
Weekly Monday, Wed. & Friday, 10:30 a.m., Pediatric Department.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 a.m., Contact: J. Kipp Charlton, M.D.
ECG Conference
Weekly Tuesday, 12:30 p.m., Pediatric Department.
Medical Grand Rounds
Weekly Wednesday, 8 a.m., 1st Floor Conf. Room.
Visiting Professor Formal Presentation
Weekly Thursday, 8 a.mM., PIMC
Visiting Professor Informal Presentation
Weekly Thursday, 9:30 a.m., 1st floor Conf. Rm.
Visiting Professor Formal Presentation
Weekly Thursday, 12:30 p.m., PIMC
Nephrology Conference
Weekly Fridays, 12:30 p.m., Pediatric Department.

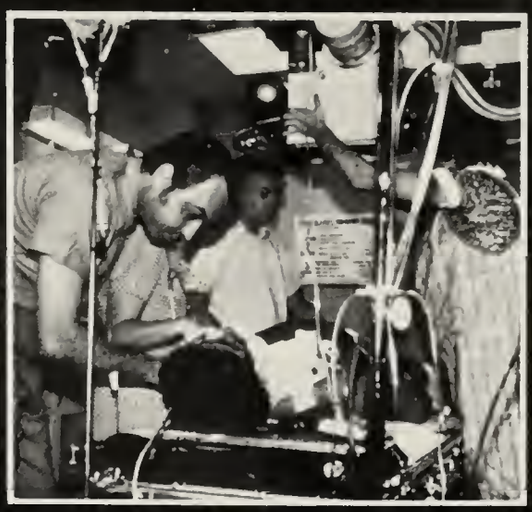
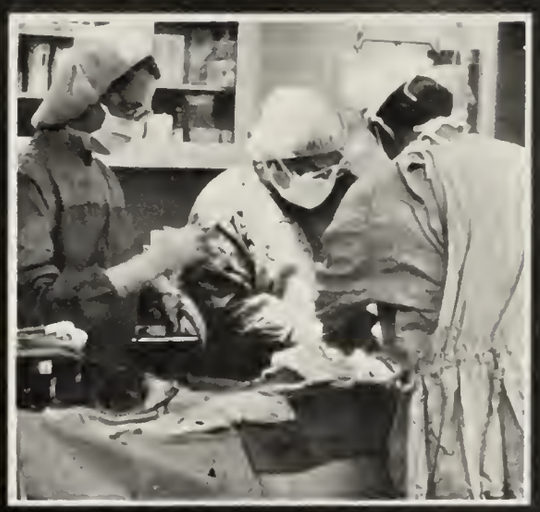
ST. JOSEPH'S HOSPITAL (THMEP) TUCSON

350 N. Wilmot Road, Tucson, AZ. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.
Family Practice Department Meeting
3rd Tuesday, 12 p.m., Contact: Wm. Moteforte.
Ophthalmology Morbidity/Mortality Conf.
4th Tuesday, 12:15 p.m., Contact: Kim Sowards.
Current Concepts in Medicine
Weekly, Tuesday, 12 p.m., Auditorium.
Hematology-Oncology Conference
Last Wednesday, 12:15-1:15 p.m., Contact: S. Salmon, M.D.

ST. MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.
Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 a.m., Century Rm. A., Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Emergency Medicine Lectures
Weekly Thursday, 8 a.m., Century Room A.
Mental Health Update
1st Friday, 11:30-1:00 p.m., Century Rm. A.
Cardiology Conference
Weekly Friday, 8:00-9:00 a.m., Century Rm., Contact: Anthony Forte, M.D.

Our trauma team . . . the fine line between life and death.

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993-8500

John C. Lincoln Hospital
Trauma Center & Emergency Services
9211 North 2nd Street - Phoenix
943-2381

Paradise Valley EmergiCenter
4232 East Cactus Road
996-6720

ST. LUKE'S HOSPITAL MEDICAL CENTER

525 North 18th Street, Phoenix, AZ, Contact: Gerald L. Hansbro, M.D.

Cardiac Conference
Weekly Monday, 12:15 p.m., Auditorium
Surgery Conference
1st Tuesday, 12:15 p.m., Auditorium
Emergency Medicine Conference
1st Wednesday, 12:15 p.m., Auditorium
Cardiovascular-Thoracic Record Review
3rd Wednesday, 12:15 p.m., Auditorium
Psychiatry Conference
3rd Thursday, 7 a.m., Auditorium
Combined Medical General Practice Conference
1st Friday, 12:15 p.m., Auditorium
Toxicology Grand Rounds
2nd Friday, 7:30 a.m., Auditorium
Ophthalmology Conference
1st Saturday, 8:30 a.m., Auditorium.

TUCSON MEDICAL CENTER (THMEP)

301 E. Grant Road, Tucson, AZ 85716.
ontact: Eric G. Ramsay, M.D. Approved for ategory 1 credit.
ardiology Conference
eekly Monday, except last, 12 Noon, ontact: J. Fernadez, M.D.
ermatology Conference
h Monday, 5:00 p.m., Contact: R. Miller, .D.

Endocrinology Conference
Last Monday, 12 p.m., Contact M. Parker, M.D. **Perinatal Conference**
2nd Monday, 7:30 p.m., Contact: J. Lohman, M.D.
Chest Conference
3rd Tuesday, 12 noon, Contact: William Nevin, M.D.
Hematology Conference
4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Infectious Disease Conference
2nd Tuesday, 12 noon, Contact: B. Friedman, M.D.
Nephrology Conference
1st Tuesday, 12 noon, Contact: Dr. Seltzer.
Orthopedic Conference
1st Tuesday, 8:00 p.m., Contact: Jay Katz, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12:30 p.m., Contact: Dr. Lightner.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 p.m., Contact: L. Buckels, M.D.
Clinical Pathology Conference
Last Wednesday, 8:00 a.m., Contact: Dr. Fuchs.
Family Practice Meeting
2nd Wednesday, 12:30 p.m., Jan., April, July, & Oct. Contact: C. Mangelsdorf, M.D.
Medical Conference
Weekly Wednesday, 8:00 a.m., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 noon, Contact: H. W. Buschsbaum, M.D.

Dermatology Conference
1st Thursday, 12 noon, Contact: R. Friedman, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 p.m., Contact: N. Komar, M.D.
Tumor Conference
Weekly Thursday, 12 noon, Contact: Dr. Hastings.
G—I Conference
Weekly Friday, 12 noon, Contact: C. Krone, M.D.
Interhospital Nuclear Medicine Conference
Weekly Friday, 7:15 a.m., Contact: S.V. Hilts, M.D.
OBGYN Conference
1st Friday, 7:30 a.m., Contact: P. Evans, M.D.
OBGYN Pathology Conference
3rd Friday, 7:30 a.m., Contact: R. Spark, M.D.

Surgical Department Conference
Weekly Saturday, 7:30 a.m., Contact: Dr. Hastings, TMC.
Surgical Grand Rounds
Weekly Saturday, 9:00 a.m., UAHSC-Room 5403, Contact: Dr. Norton.

PHOENIX VETERANS ADMINISTRATION MEDICAL CENTER

7th Street and Indian School Road, Phoenix, AZ 85012. Contact: Alfred Heibrumn, M.D. Approved for Category 1 credit.
GI X-Ray Clinical Correlation Conf.
1st & 3rd Monday, 1 p.m., Rm. C435, Contact: Drs. Sanowski, & Grobe.

Medical/Surgical GI Conference
1st & 3 rd.Mondays, 3 p.m., Rm. 3134, Contact: Dr. Kozarek, Ext 413. Dr. Mertz, Ext 493.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Monday, 4 p.m., Rm. 3134, Contact: Dr. Mertz, Ext 493.
GI Pathology Conf.
2nd Monday, 1 p.m., Path Conf. Rm, Contact: Drs. Scowloft & Lenard.
Vascular Conference
2nd Monday, 3 p.m., Room 3134, Dr. Cintora, Ext 419
Surgery/Pathology Conference & Tissue Review
1st & 3rd Tuesday, 4 p.m., Room 2128, Contact: Dr. Landard
GI Grand Rounds
Weekly Tuesday, 1 p.m., Contact: Drs. Sanowski, Kozarek & Partyka
Cancer Symposium
Weekly Wed., 1 p.m., T-5, Contact: Dr. Byrne, Ext. 426.
Housestaff Pulmonary Disease Teaching Conf.
Weekly Wed., 3-4 p.m., Contact: Dr. Rohwedder, Ext. 388.
Surgical X-Ray Conf. & Bedside Teaching Rounds
Weekly Wednesday,4 p.m., Rm. 3134. Contact: Dr. Mertz, Ext. 493.
Medical Surgical Chest Conference
1st & 3rd Thursday, Contact: Dr. Rohwedder or Dr. Gill.
Cardiology Conference
2nd Thursday, 1 p.m., Rm. T-5.
Medical Service Grand Rounds
Weekly Friday, 11 a.m., Contact: Dr. Zeller
GI Resident Conference
Weekly Friday, 1:00 p.m., D446, Contact: Drs. Grobe, Phelps, Sanowski and Kozarek.
Surgical Journal Review & Videotape Session
Weekly 1 p.m., Room 3134.

PRESCOTT VETERANS ADMINISTRATION HOSPTIAL MEDICAL CENTER

Prescott, Arizona 86313. Contacts listed below. Approved for category 1 credit.
Medical Lectures
1st & 2nd Thursday & Weekly,Fridays, 1:30 p.m., Bldg. 28. Contact: J. W. Fitzharris, D.O.
GI Rounds
Third Thursday, 10 a.m.-2:30 P.M., Contact: J. W. Fitzharris, D.O.
Grand Rounds
Weekly Thursdays, 11 a.m., Bldg. 107. Contact: J. W. Fitzharris, D.O.

TUCSON VETERANS ADMINISTRATION HOSPITAL & MEDICAL CENTER (U of A Tucson)

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.
Medical Surgical Chest Conference
Weekly Tuesday, 2 p.m., Contact: Dr. Young
Medical Grand Rounds
Weekly Wed., 12-1:00 p.m., VA Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 p.m., Contact: Dr. Putnam
Endocrinology Seminar
1st, 3rd, & 5th Thursday, 12-1:00 p.m., Rm. N318, Contact: Dept. of Internal Medicine.
Grand Rounds
Weekly, Thursday, 11 a.m., Bldg. 107, Contact: J. Fitzharris, D.O.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 noon.
Neurology Grand Rounds
Weekly Friday, 12 p.m., Contact: Dr. Sibley.

YUMA REGIONAL MEDICAL CENTER (U of A, Tucson/ArMA)

2400 Avenue A., Yuma Az 85364. Contact: Alan Winfield, M.D. Approved for category 1 credit.

Radiology Conference
1st Tuesday, 7:00 a.m.
Operation Outreach
2nd Tuesday, 6:30 p.m.
Pathology Conference
4th Tuesday, 7:00 a.m.
Operation Outreach
2nd Wednesday, 7:00 a.m.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.
Anesthesiology Board Review Conference
2nd & 4th Monday, 4-5 p.m., AHSC Dining Rm. C&D, Contacts: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4-5 p.m., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 a.m., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4-5 p.m., AHSC Dining Room, C&D, Contacts: Drs. Otto & Zehngut.
Cancer Center Tumor Board Seminar
3rd Tuesday, Monthly, 12-1 p.m., HSC Auditorium, Contact: Cancer Center
Cardiac Catheterization Conference
Weekly Friday, 4:00 p.m., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 a.m., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 p.m., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 p.m., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 p.m., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Rounds
Weekly Wed., 11:30 a.m., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12-1 p.m., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 p.m., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 a.m., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 p.m., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 p.m., Rm. 6505. Contact: S. Salmon, M.D., Dept. of Internal Medicine.
Medical Grand Rounds
Weekly Wed., 12-1 p.m., AHSC, Contact: Dr. J. Smith.
Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 a.m., AHSC, Contacts: Drs. Hughes & Alcorn
Neuromuscular Disease Conference
Weekly Friday, 11:30 a.m., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 a.m., Rm. 5120, UAHSC.
Neuroradiology Conference
Weekly Thurs., 5:00 p.m., Contact: Dr. P.C. Christenson
Neuromuscular Journal Conference
2nd & 4th Thursday, 7-9 p.m., Contact: Dr. Stern.
Neuropathology Case Review
Weekly Friday, 8:30 a.m., AHSC, Contact: Dr. P. Johnson.
Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 a.m., AHSC, Contact: Dr. C. Bamford
Nuclear Medicine
Weekly Thursday, 7:30 a.m., AHSC Radiology Conference Rm.
Ophthalmology Grand Rounds
Weekly Friday, 8 a.m., AHSC, Contact: Dr. H. Cross
Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 p.m., AHSC Contact: Dr. H. Cross
Orthopedic Rounds
Saturday, 8:00 a.m., Contact: Dr. Peltier.
Pain Conference
3rd Monday, 4-5 p.m., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.
Pathology Seminar
Weekly, 4:30-5:30 p.m., AHSC, Rm. 5120. Contact: Dr. P. Finley
Tucson Pathologist Conference
1st Monday, 7:30 p.m., AHSC, Contact: Dr. A. R. Graham.
Pediatric Grand Rounds
2nd & 4th Tuesdays, 12 p.m., AHSC, Contact: Dr. H. Thompson.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 a.m., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 a.m., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 a.m., Contact: Dr. Philip Walson.
Psychiatric Grand Rounds
Weekly Wednesday, 5:30 p.m., AHSC, Rm. 8403, 5th Floor Auditorium
Psychiatric Monthly Case Conference
2nd Friday, 7:30 a.m., Contact: Dr. Alan Levenson, Palo Verde Hospital
Pulmonary Rounds
Weekly Friday, 11:30 a.m., Contact: Dr. Benjamin Burrows.

Chest Radiology
Weekly Monday, 5-6 p.m., Rm. 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 a.m., AHSC, Contact: Dr. Christenson

Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 a.m., AHSC, Rm. 0655

Radiology Interesting Case Conference
Weekly Thursday, 12:00 noon, AHSC, Contact: Dr. Freundlich

Gastrointestinal Radiology
Weekly Tuesday, 5-6 p.m., AHSC, Contact: Radiology Dept.

Radiology-Rheumatology Conference
Weekly Thursday, 7:45 a.m., UAHSC, Library Rm.1535C.

Uroradiology (Genitourinary Radiology)
2nd & 4th Mondays, 7:30- 8:30 a.m., AHSC, Contact: Dept. of Radiology

Renal Pathology Conference
1st & 3rd Thurs., 11:30 a.m., Contact: Dr. Nagle.

Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 noon, AHSC, Contact: Dr. A. Greensher.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 a.m., Contact: Dr. Wangensteen.

Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.

Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 a.m., Contact: Dr. M. Silverstein.

Toxicology Conference
Weekly Tuesday, 8:00 a.m., Contact: Dr. Keith Likes
Section.

Tucson Ultrasonography Group
Weekly Wed., 4:30 p.m., AHSC, Contact: Dr. I. Freundlich.

General Urology Conference
Weekly Tuesday, 12:00 noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

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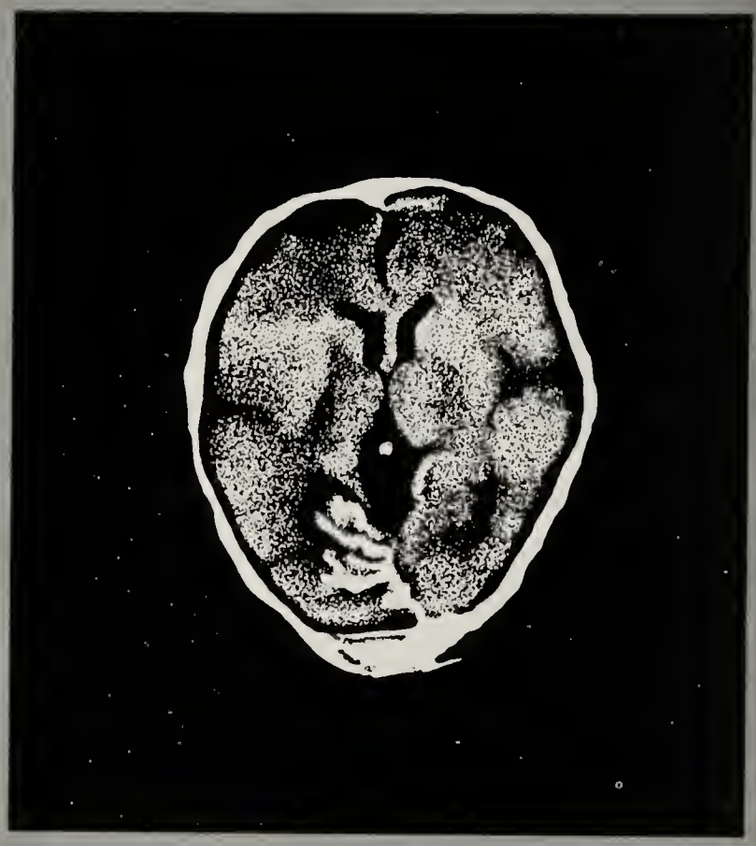
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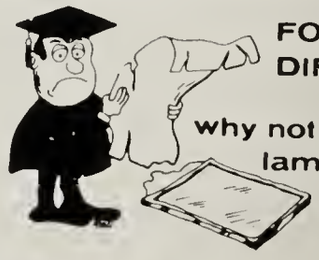
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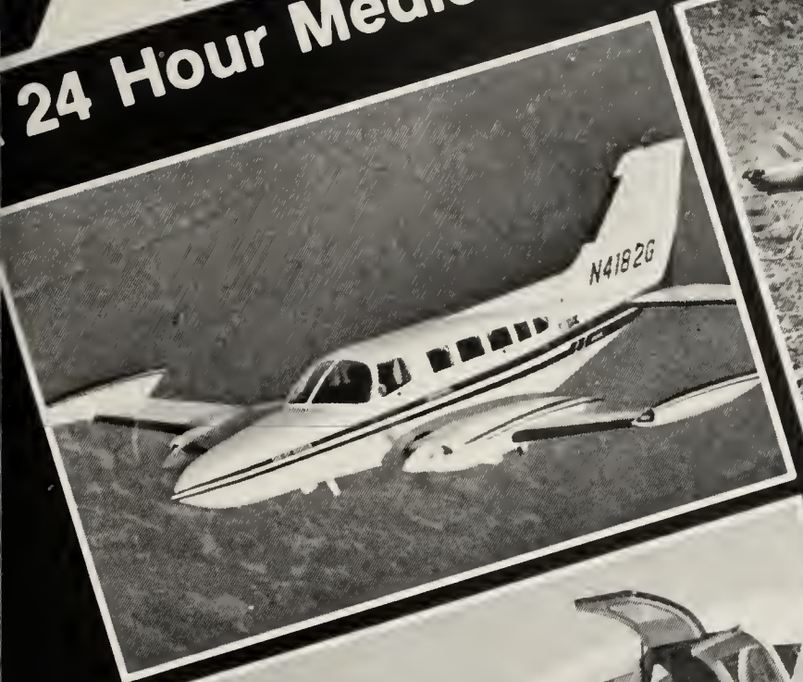
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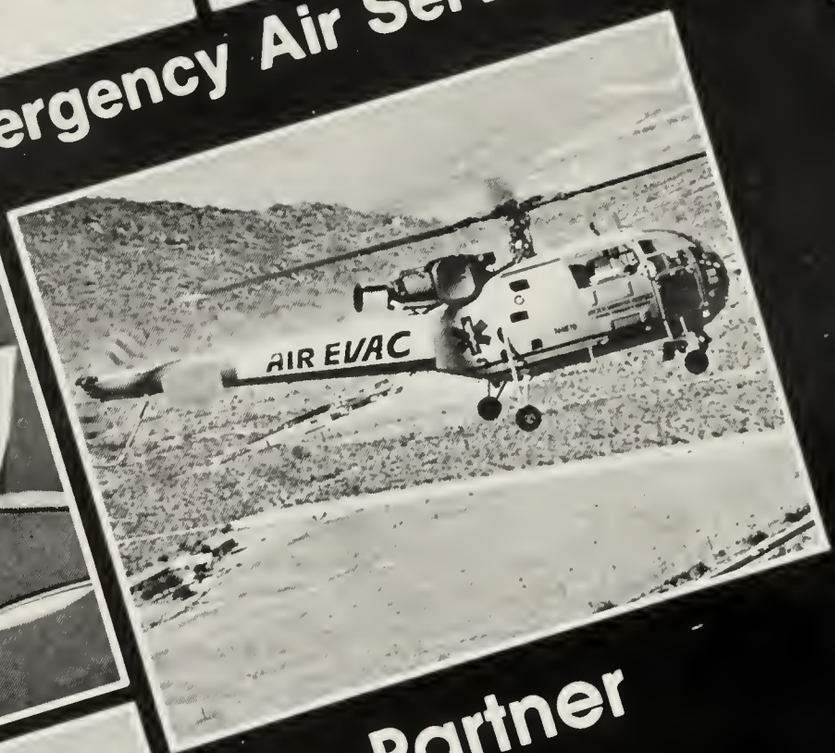
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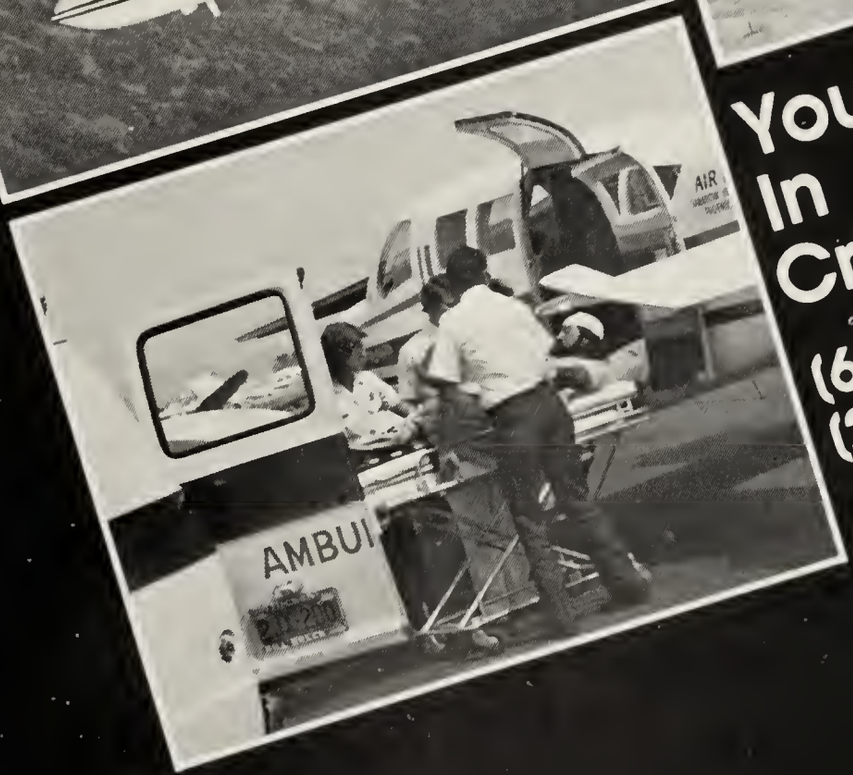
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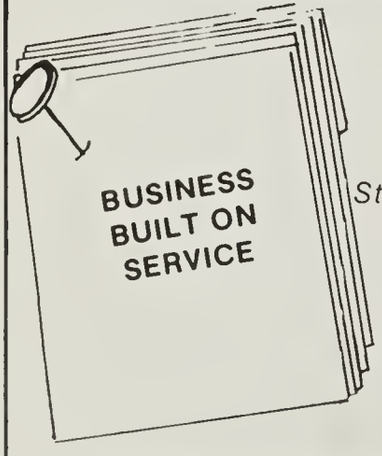
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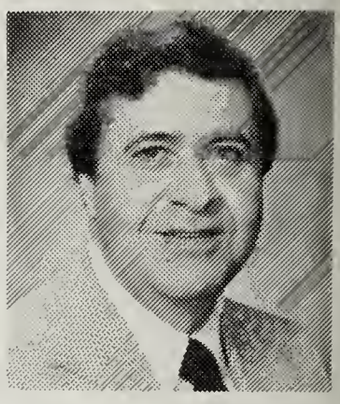
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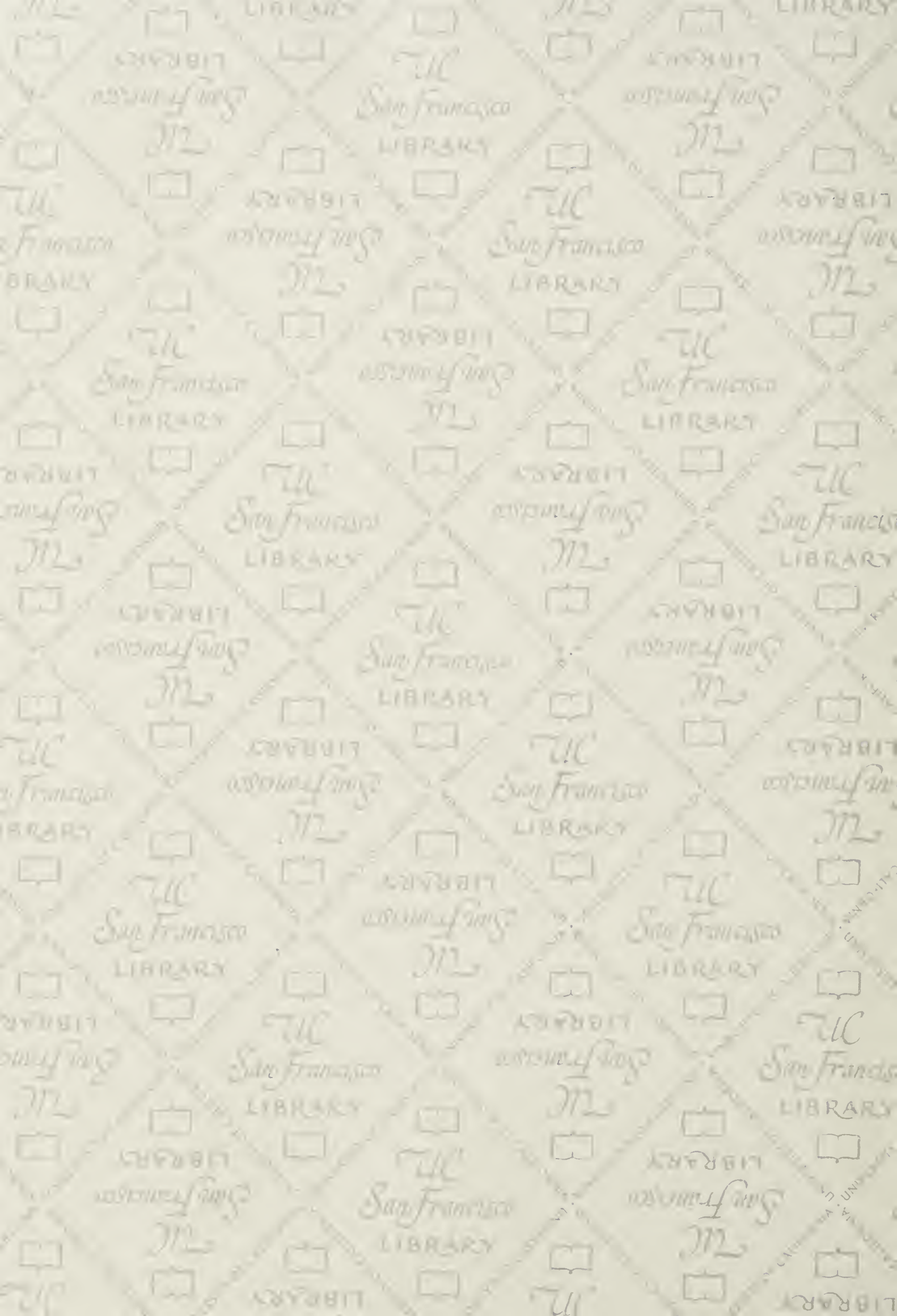
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